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THE MEDICAL
DISEASES OF CHILDREN

THE MEDICAL DISEASES OF CHILDREN

BY

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PREFACE

IN the following pages I have endeavoured to write a short but complete account of the Medical Diseases of Children in accordance with modern views. I have throughout assumed a working knowledge of the diseases of adults, and have tried to put the reader in the way of acquiring a similar grasp of the subject of pædiatrics.

My aim has been to produce a book of teaching value, and its contents, and the order, length and phraseology of its articles, have been arranged with this end in view. The needs of the student and practitioner, as I have experienced them in teaching children's diseases, have been chiefly considered, but I have not hesitated to touch upon some of the more important of the problems as yet unsolved in such a way as I hope may not be without value to those who are interested in the advancing line of clinical medicine.

The arrangement of the book, particularly with regard to the section on the infective diseases, needs a word of explanation. Probably the time will come when we can classify all the infective processes mainly according to the infecting organism, and we shall speak of the pneumococcal, staphylococcal, various streptococcal and other infections as diseases, in the same way that we have for long spoken of tuberculosis, and more recently have learnt to speak of rheumatism. In the case of adults this is not possible as yet, but the infections of children lend themselves much more easily to this method of description, and I have felt it right to endeavour to adopt it here. Thus I have described in order all the manifestations of the pneumococcal infection, then all those of the tuberculous, rheumatic and other infections. This arrangement, which is I think theoretically sound, is also of practical value in that it emphasizes the great tendency towards generalization which is shown by all bacterial infections in children.

In order to increase the teaching value of my book, I have included as many useful illustrations as possible. The disadvantage which has been urged against the use of photographs, namely, that they over-emphasize the obvious to the neglect of signs which are early and ill-marked, is I am sure, more than counterbalanced by their power to teach quickly, clearly, and, within certain limitations, accurately.

It is my pleasant duty to acknowledge my indebtedness to those who have helped me in various ways with the production of this book.

First, I must express my thanks to the physicians at the Hospital for Sick Children, Great Ormond Street. To the teaching I received during the years that I was Medical Registrar and Pathologist there, I owe more than I can well acknowledge. I am under further obligation to them for permission to make use of those photographs which I took for the records of their cases. These, together with some of my own cases, kindly taken by residents at the Paddington Green and St. Mary's Hospitals, form the majority of the illustrations of this book.

To Dr. David Lees and my colleagues at Paddington Green I am indebted for various photographs. Professor R. Stockman has kindly sent me a tracing illustrating an important point in the pharmacological action of sodium salicylate, part of which is shown in *Fig. 32*.

Some of the illustrations have appeared elsewhere. Those of a case of adrenal sarcoma are from photographs which I took for Dr. R. Hutchison's article on that condition which appeared in the first number of the *Quarterly Journal of Medicine*. The illustrations of rheumatic myocarditis (*Figs. 61 and 62*), and of amyotonia congenita (*Figs. 96 and 97*) appeared in two articles by Dr. Carey Coombs in the *British Medical Journal*. *Fig. 22* is after an illustration in Dr. Still's *Common Disorders and Diseases of Childhood*. The two illustrations of precocious obesity appeared in Dr. Leonard

Guthrie's article on that subject in the Transactions of the Clinical Society. Figs. 56 and 66 are after charts appearing in papers by my colleague Mr. D. C. L. Fitzwilliams and my friend Dr. H. Moreland McCrea in the *Lancet* and the *Practitioner* respectively. To these authors, and to the publishers and editors of the publications mentioned, I am much indebted. In the case of Figs. 70 to 74 and Figs. 61 and 62 I have further to acknowledge the kind loan of the original blocks. Fig. 24 is from a paper of my own, and is published by the kind permission of the Registrar of the Medical Society of London.

To my friend, Dr. Leonard Parsons, of Birmingham, I am particularly indebted. For the photographs which he has provided (numbering nearly a dozen), for the care and time he expended on the first proofs of my book, and for many suggestions and improvements in the text, I tender him my best thanks.

Dr. Carey Coombs has kindly given me the benefit of his criticism of the chapters relating to acute rheumatism and rheumatic heart disease, subjects in which we are mutually interested. Dr. Moreland McCrea has aided me in reading the revised proofs.

Miss E. A. B. Wilson, Lady Almoner to St. Mary's Hospital, has kindly revised the second appendix, which I trust will be found of service to practitioners in dealing with children of the poorer classes.

Finally, my thanks are due to the publishers, Messrs. John Wright & Sons Ltd., for the care they have expended upon the production of the volume, and for the courtesy, help and advice by which they have so materially reduced the burden of authorship.

REGINALD MILLER.

London, W., April, 1911.

CONTENTS

PAGE
1

SECTION I.—THE EXAMINATION OF CHILDREN

SECTION II.—THE DEVELOPMENT AND FEEDING OF CHILDREN.

I.—PHYSICAL AND MENTAL DEVELOPMENT	18
II.—THE FEEDING OF HEALTHY CHILDREN	25
III.—INFANT FEEDING IN DIFFICULT CASES	45
SUMMARY	50

SECTION III.—CONSTITUTIONAL DISEASES.

I.—RICKETS	61
II.—INFANTILE SCURVY	73
III.—DIABETES MELLITUS	80
IV.—ACIDOSIS AND ACID INTOXICATION	86

SECTION IV.—INFECTIVE DISEASES.

I.—THE PATHOLOGICAL INFECTION	96
II.—THE TUBERCULOUS INFECTION	110
III.—THE RHEUMATIC INFECTION	131
IV.—RHEUMATOID ARTERITIS	150
V.—ERYSIPLAS NEONORI	151
VI.—DIPHTHERIA	155
VII.—THE MENINGOCOCCAL INFECTION	164
VIII.—TYPHOID AND ALIRED INFECTIONS	185
IX.—THE GONOCOCCAL INFECTION	203
X.—THE DIPHTHERIAL INFECTION	207
XI.—ACUTE POLIO-ENCEPHALOMYELITIS	209
XII.—THE ACUTE INFECTIONS FEVERS	222

III Scintilla; (ii) Measles; (iii) German Measles; (iv) Diph-
theria; (v) Whooping-cough; (vi) Chicken-pox; (vii) Mumps.

SERIES F.—DISEASES OF THE DIGESTIVE SYSTEM. 117

I.—DISEASES OF THE MOUTH	455
II.—DISEASES OF THE SALIVARY GLANDS	448
III.—DISEASES OF THE OESOPHAGUS	459
IV.—DISEASES OF THE STOMACH	454
V.—DISEASES OF THE INTESTINES	475
VI.—DISEASES OF MECKEL'S DIVERTICULUM	705
VII.—INTESTINAL PARASITES	500
VIII.—DISEASES OF THE PERITONEUM	460
IX.—DISEASES OF THE LIVER	472

SERIES FZ.—DISEASES OF THE RESPIRATORY SYSTEM.

I.—DISEASES OF THE NOSE	424
II.—DISEASES OF THE TONSILS	425
III.—DISEASES OF THE PHARYNX	428
IV.—DISEASES OF THE LARYNX	432
V.—DISEASES OF THE LUNGS	436
VI.—DISEASES OF THE PLEURA	460

SERIES FIZ.—DISEASES OF THE CIRCULATORY SYSTEM.

I.—CONGESTIVE CARDIAC ANOMALIES	464
II.—ACQUIRED HEART DISEASE	467
a) Rheumatic Heart Disease; b) Malignant Endocarditis; the Disorders of Cardiac Rhythm	
III.—DISEASES OF THE ARTERIES	480

SERIES FIII.—DISEASES OF THE GENITOURINARY SYSTEM.

I.—THE URINE IN CHILDHOOD	490
II.—DISEASES OF THE KIDNEYS	491
III.—DISEASES OF THE BLADDER	495
IV.—DISEASES OF THE GENITAL ORGANS	496

CONTENTS

xi

Series IX.—DISEASES OF THE SPLEEN, BLOOD, AND GLANDS.

I.—DISEASES OF THE SPLEEN	411
II.—DISEASES OF THE BLOOD	414
III.—DISEASES OF THE GLANDS	421
(i) Diseases of the Lymphatic Glands; (ii) Diseases of the Spleen; (iii) Enlargement of the Thymus.	

Series X.—DISEASES OF THE NERVOUS SYSTEM.

I.—MENTAL DEFICIENCY	434
(i) Congenital Idiot; (ii) Acquired Idiot.	
II.—FUNCTIONAL NERVOUS DISEASES	445
III.—ORGANIC NERVOUS DISEASES	471
(i) Congenital Nervous Diseases; (ii) Functional Nervous Diseases; (iii) Acquired Nervous Diseases.	

Series XI.—DISEASES OF THE BONES AND MUSCLES.

I.—DISEASES OF BONES	485
II.—DISEASES OF MUSCLES	500

APPENDIX A.

DISTRICT AND THERAPEUTIC MEASURES.

APPENDIX B.

SOCIETIES, INSTITUTIONS, ETC., AMONG DEAF-MUTED CHILDREN.

INDEX.

LIST OF ILLUSTRATIONS

ADRENAL SARCOMA:—	158
Appearance of head at death	163
Asymmetry of skull, displacement of, and hæmorrhage within it	174
Metastatic deposits on skull-cap	175
Tumours forming in forehead and external to eye	174
View of abdominal tumour	174
BOXES AND MUSCLES, DISEASES OF:—	
Achondroplasia	220
Aristiaia congenita	215
Cleidocranial dysostosis: skull-cap	217
Myoarthry: pseudo-hypertrophic	218
Oxycephaly	226, 220
BRONCIA ESTHESIA (consecutive):—	
Chart showing age-incidence	310
BREATHING:—	
Diagram representing forms of grouped or rhythmic breathing	10
EMPHYSEMA:—	
The "old man chest" of	155, 150
EMPHYSEMA, pointing in:—	101
EMPHYSEMA, torusoid	155
HEART DISEASE:—	
The cladding of fingers	294
Method of applying ice-bag to	277
Mitral stenosis: the stages of development	283
Rheumatic myocarditis: the histological changes in	171
Rheumatic pericarditis: showing extreme pallor of basal area	174
HUSCHKE'S DISEASE	250, 260, 301
HYDROCELE:—	
The characteristic sign of	100

LIST OF ILLUSTRATIONS

xi

HYPERTROPHIC PYLORIC STENOSIS:—

Abdominal viscera post mortem	262
Stomach showing extreme constriction	263
Gastric peristalsis	267
Hypertrophy of pyloric and gastric walls	265

INFANT FEEDING:—

Apparatus for home sterilization of milk in bottles	11
Apparatus for preparation of humanized milk	516
Hour-shaped feeding-bottle	10
Tube for centrifuge in estimating percentage of fat in milk	24
Weight chart	16
The same, showing progress of an infant on sterilized milk	20
Weight chart of an infant with curd- and fat-indigestion	11

INTOXICATION:—

Chart showing the age-incidence during first year	101
---	-----

LYMPHADENOMA:—

Showing enlargement of spleen and glands	422, 423
--	----------

MENINGITIS, EPIDEMIC MENINGOCOCCAL:—

Purpuric rash	202
---------------	-----

MENINGITIS, POSTERIOR BASE:—

Showing opisthotonos, rigidity, and emaciation	697
Brain, showing pus distribution	599

MENINGITIS, PNEUMOCOCCAL:—

	111
--	-----

MENTAL DEFICIENCY:—

Cretinism	436, 446, 441
Mongolian imbecility: Facies	456, 457
— — — Hand with short, incurved little finger	457

NERVOUS SYSTEM, DISEASES OF:—

"Cerebellar tilt" of head	492
Cerebral sclerosis showing gliosis	476
Congenital cerebral dysplasia: cross-legged progression	475
"Hypertrophy of pons": diffuse posterior glioma	101
Method of eliciting Chaddock's sign	454
Paralysis (Erlé's) from acute poliomyelitis	218
Peroneal type of muscular atrophy	481
Tetany	449, 451

PETRIE'S:—

Funnel-chest following	248
Subconjunctival haemorrhage	248

PNEUMONIA, PRIMARY —	
Chart showing signs and symptoms	38
Chart of protracted case	39
PERICARDIAL ORGANS	405, 447
PERITONITIS, PRIMARY —	
Temperature chart of	43
RICKETS —	
"Cat's back" posture	97
Deformed chest, posterior aspect of	94
Deformities of chest, clavicles, and long bones	93
Deformity of legs (a common)	97
"Hot-crook-ban" head	95
Rickety head	95
Rickety hypotonia	98
Rickety and pigeon chests (posterior view)	5
RHEUMATIC DYSMETABOLISM —	
Cardiac rheumatism, chart of a case of	130
Clonus, extreme hyperextension of fingers in	99
Residual clonus, myocarditis, and aneurism chart of a case of	131
Rheumatic nodules	145
Salicylates, blood pressure bearing to the effects of	135
RHEUMATOID ARTHRITIS —	
Joint changes in	225, 374, 375
SCURVY —	
Hæmorrhage causing closure of eye	88
Hæmorrhage around eye	79
Hæmorrhage into gums round erupting incisor teeth	75
Subperiosteal hæmorrhage and new bone formation	78
Swelling of thigh due to subperiosteal hæmorrhage	79
SCURVY, DYSMETABOLISM —	
Labial fissures and salivary gland of nose	181
Liver, showing peritheliosis, fibrosis, and hypertrophy	186
Spleenitis, splenitis	182
Typical "scurvy" leg	83
TEMPERATURE, PNEUMONIA —	
Showing enlarged alveoli and unfolded alveoli	319
TEMPERATURE, PNEUMONIA —	
Showing enlarged interlobular gland	115

LIST OF TABLES

TABLE	PAGE
1.—Age-incidence in some diseases of children	7
2.—Diagnostic points of value in the examination of cerebrospinal fluid	16
3.—Dates of eruption of teeth	20
4.—Important dates in the mental development of a child	22
5.—Comparison of constituents of human and cow's milk	23
6.—Scheme for breast-feeding	26
7.—Comparison of human milk with cow's milk, undiluted and in various dilutions with water	33
8.—Dilutions of milk (approximate) to be used at various ages	35
9.—Percentage of fat in cream and water mixtures	37
10.—Schedule for the feeding of infants on diluted cow's milk	39
11.—Wellford & Son's humanized milk preparations	47
12.—The Express Dairy Co.'s humanized milk preparations	48
13.—Composition of "Gloss," undiluted and diluted	48
14.—The composition of cow's, don's, and human milks compared	49
15.—The composition of cow's milk, whey, and human milk compared	49
16.—"Humanoid" compared with human milk	54
17.—Infant foods intended as substitutes for fresh cow's milk, arranged in order of fat percentages	55
18.—Infant foods intended as additions to cow's milk	55
19.—Showing usual differences between posttesticular meningitis and tuberculous meningitis	147
20.—Some dates in connection with the acute infectious fevers	223
21.—The chief points in the differential diagnosis between scarletina, measles, and rubella	228
22.—Showing differences between hypertrophic pyloric stenosis and dyspepsia (pyloric spasm) of infants	272
23.—Differential diagnosis between mononucleosis and erythema	358

THE MEDICAL DISEASES OF CHILDREN.

SECTION I.

THE EXAMINATION OF CHILDREN.

THE EXAMINATION OF CHILDREN.

THE child in its diseases is no mere miniature adult. Upon this fact rests the necessity for the special study of disease in children. To the lay it is a surprise that any such necessity can exist, but to the young practitioner the fact becomes painfully clear, for he finds that a good working knowledge of morbid conditions in adults is quite compatible with a feeling of melancholy helplessness when he is confronted by a sick child.

In such a book as this we may therefore well open with a consideration of those points which are the real or imaginary difficulties of the subject, mentioning some of the chief differences between disease as it occurs in children and in older subjects.

Many conditions occur only in children, while more are very much more frequent in them than in adults. On the other hand, a large number of the diseases of adults are not seen in children. When, however, a disease which is common at a later age occurs in childhood, it is often considerably modified in its characteristics, especially where the patient is under the age of eight or ten years. After this period diseases begin to show a clinical picture similar to that seen in adults, e.g., such infections as the rheumatic or pneumococcal.

Even dealing solely with children the symptoms and dangers of a disease vary considerably with the age of the patient.

Few facts are more worthy of remembrance than the tendency towards generalisation which bacterial infections manifest in children. Subject to the age incidence of the infection, the younger the child the greater is the danger of generalisation, while in all it occurs more readily than in adults. This is well seen in the three great bacterial diseases, the tuberculous, pneumococcal, and rheumatic infections.

The feeding of infants in sickness is a science which, in its difficulties and dangers, has practically no counterpart in the medical treatment

of adults, with whom in no disease is a slight change in the diet thought with so great possibilities of good or evil as in the case of an infant.

But while the regulation of food is literally often a matter of life or death, the dosage of drugs is of much less importance, and in some minds seems to be a much exaggerated difficulty. There are, it is true, some drugs which are particularly dangerous in children, but these are few in number, while others may be given with greater freedom than in adults. These matters are discussed elsewhere (Appendix A).

The impossibility of obtaining a detailed description of symptoms from a child is only occasionally a cause of difficulty in diagnosis. As a rule, it is amply compensated for by the clearness with which various forms of disease leave their imprint upon the child.

The chief difficulty experienced in treating children lies in the fact that the mind, trained to observe in their true perspective the details which go to form the picture of disease in adults, finds itself out of focus as it were for the signs as seen in sick children. A symptom, rare in an adult, may be the rule in children; what is serious in grown-up people may be comparatively trivial in a child, or vice versa. The greatest difficulty of all, perhaps, is that a symptom which in an adult suggests a certain set of conditions, in a child may point to some totally different disease. For instance, stridor in an adult may suggest narrowing of the air-passages by pressure, growth, or cicatrization, while in a child we should first consider such possibilities as retropharyngeal abscess, laryngeal diphtheria, or a foreign body impacted in the larynx or oesophagus. To get the mind again into focus, to learn what is common, what is to be expected, the significance of this symptom, the value of that sign, these constitute the groundwork of a knowledge of children's diseases.

THE EXAMINATION.

In approaching the subject, one is haunted by the well-known phrase, "Let us first endeavour to acquire the confidence of our little patient." Although a matter of importance, yet there is little that need be said on this score, for most men can get on perfectly well with children for the short time necessary for a medical examination. Each must adopt whatever means he finds himself to possess by which he can please children; and when all is said, the difficulties which arise in connection with children's diseases are concerned with the interpretation rather than the eliciting of signs.

It is only common sense to talk to a child for a little while before beginning the examination, to be gentle, to use warm hands (an important point), to let the child play with the stethoscope, or have its use explained to him if he be nervous. Much patience is often required; any sign of irritation or displeasure on the part of the examiner may produce disastrous results.

Of all the resources at command none is more effective than a sympathetic voice. Even an infant should be talked to while being examined. To allay a child's fears and to distract its attention, nothing is so useful as a conversation. Questions may be asked about other members of the family, lessons, teachers, pets, and a host of other matters, avoiding such as can be answered by a simple "yes" or "no," which may be indicated by movements of the head. Naturally, during the early days of a child's stay in hospital, any references to its mother are best omitted. By getting the child to speak, regular respiratory movements of the chest, and the muscular relaxation necessary for the testing of tendon reflexes or for the palpation of the abdomen, are satisfactorily obtained.

Some of the methods by which special signs may be conveniently elicited, are given later.

Most small infants are best examined in their mother's arms, lying on her lap or, for examination of the back, looking over her shoulder. Many, especially at the age of nine or ten months, will be quiet if they are allowed to sit up, but cry loudly if while they are put on their backs. To stroke or rub the top of an infant's head with the palm of a warm hand is often conducive to peace. It is frequently wise to leave a child undressed before it is seen by the doctor, as it is then less liable to start its indignance upon him.

A nervous child may require much comforting and many explanations before it will submit to an examination. Some there are who are dragged into the consulting-room yelling and shrieking. Such behaviour is usually due to the threatnings of a foolish mother, who has pointed to the child in a loud light for many days, what may be expected to happen on its visit to the doctor if it refuse to eat its food or conform in other ways to the expected mental standard. Naturally enough, the child is convinced of the close proximity of a painful ending. The patient in such a case is never seriously ill, and the best plan is to attend to the mother, taking no notice of the child until it is time to say good-bye. This word generally releases the child considerably, and it becomes possible to explain that next time he must behave better, and that under no circumstances will he be hurt. At the next visit the child is usually easily handled.

There are some who cry because they are shy rather than frightened. These may usually be lulled by promises of some reward if they are good. In others, again, a little sternness produces quiet.

The really irritable child is a source of great difficulty. He rejects all advances of friendship, replies "No!" to all questions, says "I don't want to!" to all that he is asked to do, spurns all toys and promises of reward. Some simple little mortals are much affected by flattery, even of the most abject order, but not so the irritable child. He remains impervious. With him, then, it is best to make a rapid and gentle examination, talking to him continuously in a quiet voice; any rough handling will cause an outburst of screaming.

History of Present Illness.—In hospital practice a reliable account of the mode of onset of disease may be difficult to obtain. Symptoms may be so exaggerated as to give a very false impression. This is done partly in order that the doctor may be impressed with the seriousness of the child's condition, and partly from the romantic turn of mind of the ill-educated. Imperfect closure of the eyes during sleep is the proximate basis for a statement that the child seems "very convulsed." A slight rise of temperature or sweating of the head is spoken of as a heat that is unapproachable, or causes burning of the mother's arm as the child rests upon it. Sleep following an attack of screaming is described as causing the child to "lay like a little dead thing." It is curious to notice how unvarying are the terms with which symptoms are described by the poor, and as they are few, each phrase has to cover a number of conditions. Sometimes words are added because a previous word has suggested them, although in themselves they mean little to the speaker. This "small but very compact" may be the mother's description of her infant's condition at birth. In some cases, at least so it would appear, the mother, in order to engage and interest the doctor, collects what symptoms she recalls as having occurred in her other offspring, adds such as are suggested to her by her neighbours and by those next to whom she has sat in the out-patient department, and presents a tale of plague, pestilence, and famine which ill accords with the comparatively healthy appearance of her child.

Nevertheless, nothing is more foolish than to disregard on principle what a hospital mother may say. Fortunately, in serious cases the history of the illness is usually much more carefully and accurately given.

In a few cases, but they are really quite the exception, a diagnosis can only be made when a proper account of the onset of the symptoms is forthcoming. Particular difficulty arises in connection with congenital nervous conditions; it must be remembered that such congenital diseases may not be noticed until the child reaches the age at which it should begin to walk.

Previous History.—In the case of infants it is well to inquire into the duration of the pregnancy, the character of the labour, the condition at birth, the presence or absence of snuffles or rashes, and the method by which the child has been fed. The occurrence of past illnesses, especially of the acute specific fevers, should be noted. In inquiring for past manifestations of thrombosis, it is necessary to ask about sore throats, growing pains, and chorea, as well as rheumatic fever itself. In this connection the importance of scarlatina may be borne in mind.

Family History.—Both tuberculosis and rheumatism tend to run in families. Inquiry should be made into the possibility of direct

infection in such cases. In such common diseases there is little use in investigating further than the immediately preceding generation. Evidences of an inherited syphilitic infection should be carefully examined. A miscarriage occurring in the course of many pregnancies is of no value in diagnosis, but a history of several miscarriages followed by a premature birth, or a still-birth at full term, is one on which great stress may be laid.

In inquiring into the nervous heredity of a child, attention may be paid to such points as insanity, epilepsy, migraine, asthma, diabetes, or general nervous instability in the family. Often a conversation with the parents gives an insight into the mental condition likely to be found in the child.

Hygienic Conditions.—The hygienic conditions at home, and the health of other children in the home, neighborhood, or school, may throw light upon the cause of disease and the treatment to be adopted.

Order of Examination.—The order to be observed in the examination of the various systems in a child is of necessity very varying. For the counting of the pulse and respiration rates, it is essential that the child is quiet, so that it is often well to begin with these preliminaries before the patient is uncovered. It is difficult to palpate the abdomen satisfactorily if the child is crying, and it may be wise to pass the warmed hand under the clothes and gently to palpate the abdominal organs. During this manipulation, while conversing with the child, the clothes may usually be raised without causing any trouble, and the abdomen inspected. After this, the heart may be examined, and then the lungs, for which part of the examination crying is no drawback and is often serviceable. The examination of the limbs, the lymphatic glands, and the rest of the body must not be omitted, but may be made at any convenient time. Best left until the end, is the important inspection of the mouth and throat.

Methods of Examination.—Of these, both inspection and palpation are of greater value in the examination of children than in that of adults. Many diseases may be recognized by inspection alone. Palpation is of special importance in the examination of infants. Whenever the child is touched, either in palpation or percussion, warm hands are essential. Percussion should be very light, if heavy, it may not only frighten the child, but lead to inaccuracy. If there be signs of rigidity, it is wise to lay the whole hand upon the child at first, and then to take off all but that part of the finger to be percussed. In small children it is often advantageous to use the little finger for this purpose.

Auscultation is best conducted by means of a binaural stethoscope; the wooden pattern of instrument causes much more fright. As a rule no difficulty arises from the use of the stethoscope if the child has

been allowed to see and handle it and its use has been explained. Occasionally, every time the position of the chest-piece is changed the patient flinches and squirms. This can often be overcome by allowing the fingers holding the chest-piece to come in contact with the child's skin for a moment before the hard edge of the instrument actually touches it. It is scarcely ever necessary to make use of the smaller chest-pieces which are now provided with most stethoscopes. It is advisable to warm the end of the chest-piece for a moment in the palm of the hand before applying it to the child's skin.

General Appearance.—Apart from the presence of such symptoms as anemia, rickets, jaundice, and the like, the general appearance of the child may at once give an important clue to the diagnosis of disease.

The tuberculous child is a well-recognized type. There are pallor, delicate features, long silky hair tending to descend on to the temples and cheeks, long eyelashes, and perhaps some downy hair on the upper lip or between the scapula. It is to be remembered, however, that such an appearance is not pathognomonic of tuberculosis, but is probably merely the result of a chronic toxæmia, of which one of the commonest is that due to tuberculosis. An exactly similar condition, for instance, may be seen in a case of chronic emphysema.

Again, the rheumatic child, with its pale face and slightly flushed cheeks, betrays its disease by the emotional expressions that flash across its face, the little fidgety movements of the limbs, and other signs of general nervous instability. The well-known faces of inherited syphilis, and the dull, heavy countenance with bright eyes of epilepsy, may be cited as examples of the clear way in which disease may imprint itself upon the appearance of the child.

One of the first things which has to be learned in the study of children's diseases is the recognition at sight of various diseases and symptoms. When we consider how many may be recognized at a glance—and there might be instanced cholera, primary pneumonia, acute diarrhoea, meningitis, rickets, cretinism, mongolism, and many another condition—we see at once the importance of inspection in the examination of children.

Some diseases—namely acute eczema, scurvy, intussusception, and acute poliomyelitis and encephalitis—attack only fat and apparently healthy-looking children.

Age of the Child.—Many mistakes may be avoided by remembering the commonest ages at which various diseases occur.

Just as there are differences between the diseases of adults and of young subjects, so in those of children alone, according to the age of the child, there are marked differences in the frequency and symptoms of various diseases. A knowledge of what conditions are likely to be present in children at various ages is an important aid to the study of the diseases of young subjects.

Some of the more important age incidences of disease may be tabulated here:—

DISEASE	COMMONEST AGE
Infantile hyperplastic pyloric stenosis	2nd week
Syphilitic epiphysitis	4th week
Infantile scurvy	6th month
Infantile convulsions	
Posterior basic meningitis	
Tuberculous meningitis	2nd year
Acute poliomyelitis	
Friedreich's ataxia	4th year
Pseudo-hypertrophic myopathy	5th year
Acute rheumatism	(very rare under 2)

Table 1.—AGE INCIDENCE OF SOME DISEASES OF CHILDREN.

The Skeletal System.—This may show many evidences of disease, to be appreciated by inspection and palpation.

The Skull may show the square form of rickets, or the oval shape with protruding forehead, as in hydrocephaly. Bussing and cranio-tabes may be present.

The condition of the *anterior fontanelle* in infants is of the greatest importance. It should be closed by the eighteenth month. Its closure may be premature, as in some cases of microcephaly, but more often is delayed, the result, as a rule, of rickets. At a year it should measure rather less than an inch in length and breadth. It serves for the examination of the pulse in infants. A depressed fontanelle means collapse, and is most commonly due to diarrhoea. Bulging of the fontanelle, in the absence of screaming or convulsions, is positive evidence of raised intracranial pressure, which is due in the majority of cases to acute meningitis.

The Limbs may show the well-known signs of rickets, the tender swellings of scurvy, epiphysitis or subperiosteal abscess, and many other conditions. The *hands* require examination. In tuberculous children the fingers are often awfully long and thin, in rheumatic children the hands are moist and restless. In rickets, the little finger is short and incurved; in the cystin, the fingers are short and square-ended; in achondroplasia, the hand is ray-like.

Clubbing of the fingers develops very rapidly in children, and is early recognizable. It may be dorsal, showing a shiny, smooth swelling round the bed of the nail (Fig. 100), or palmar, with increase in the size of the pad of the finger. When well-marked it is easily recognizable (Fig. 101). In its slightest form it has to be distinguished from the changes due to the sucking of fingers or the biting of nails. It is

seen in various cardiac and pulmonary conditions, in Hazell's cirrhosis of the liver, and also, in its slighter grades, in some cases of tuberculous peritonitis. It may be associated with clubbing of the toes, and occasionally of the tip of the nose.

The Chest, apart from local bulging or recession due to disease of the heart, lungs, or spine, may show abnormalities in its general configuration.

In the *rachitic chest* there is a forward bending of the costal cartilages, with falling in at the lateral aspects of the chest and of the sternum. The rachitic rosary, Harrison's sulcus, overgrowth of the margin of the costal arch, and greenstick fractures of the ribs may be present (Figs. 10 and 11). Bending of the ribs is usually first met with at the sixth costo-chondral junctions.



Fig. 1.—TRANSVERSE SECTION OF A RACHITIC CHEST.



Fig. 2.—TRANSVERSE SECTION OF A FUNNEL CHEST.

The *funnel-chest* is of a different shape. The sternum is very prominent, and the lateral walls of the thorax are straight, so that in transverse section the chest is triangular in outline. This deformity is due to the presence of obstruction to the breathing, and since it occurs more readily in the soft chest of rickets, signs of that disease may also be present.

A funnel-shaped depression is sometimes seen in the episternal region, and is usually due to whooping-cough (Fig. 40).

The Spine should be examined, especially in cases of chronic abdominal pain or of spastic paraplegia. The commonest forms of curvature are those due to rickets or tuberculous caries. In the former, the projection is rounded rather than angular, but a more important difference lies in the fact that if the child is quiet and not twisting, the projection due to rickets disappears when the patient is put on his back and raised by the feet, while the rigidity due to tuberculous disease remains. Severe deformity may be the result of acute poliomyelitis. Congenital scoliosis may be due to the presence of an extra half-vertebra.

The Digestive System.—The tongue in infants may be seen, as a rule, by gently depressing the chin, but occasionally it may be necessary

to put a little glycerin or syrup on the lips to make the child protrude its tongue. In older children who obstinately refuse to open the mouth, the lower lip may be pressed against the teeth. Another method is to hold the child's nose so that the lips have to be parted for breathing purposes, when a spatula is slipped in between the teeth. The inspection of the throat and mouth is of the greatest importance, and must never be omitted.

The teeth may show the Hutchinsonian form, the irregularity of ridges, or bevelled edges due to the habit of grinding the teeth. Carious teeth in themselves are sometimes responsible for ill-health.

Scarring round the mouth or anus may be a sign of inherited syphilis. It is to be noted that in order to be diagnostic of this disease, the scarring round the mouth should not be confined to the angles of the mouth, but be present also round the lips (*Fig. 38*).

Enlargement of the abdomen is most frequently due to abdominal tuberculosis or rickets. Distention is common, however, in bad cases of malarious or pneumonic, and is a sign of serious import. Ascites is most commonly the result of tuberculous peritonitis, less often of syphilitic hepatic cirrhosis, cardiac or renal conditions.

The liver and spleen may often be most easily examined while the child is in a sitting position. Not uncommonly these organs appear to be larger clinically than is in reality the case. The normal measurement and the causes of alteration in the size of the spleen are described in Section IX. The commonest form of enlargement of the liver in infancy is the fatty liver associated with intestinal dyspepsia.

The Stools.—The normal conditions of the stools are described on p. 24. Where the caecum of milk is not completely digested, white-fakes of curd appear in the motions. Unless rapidity in the passage of the intestinal contents gives rise to green motions. If fat is being given in too large quantities in the diet, the stools become pale or green, greasy, and loose. We may distinguish between the green or almost colourless watery stools of acute diarrhoea, the offensive grey stools of rickets or intestinal tuberculosis, and the large, undrained, porridge-like stools of morbus coeliacus. The causes of blood being passed per rectum are given elsewhere. Other abnormalities of the motions are mentioned under the various diseases in which they occur.

The Respiratory System.—In the examination of the lungs, good air entry is obtained by getting the child to talk, by which means both vocal fremitus and resonance may also be tested. Vocal fremitus is naturally feebler in children than in adults. Crying is often an aid to the examination of the lungs. The normal puerile character of the breathing must be borne in mind.

The signs of pleural effusion are given and discussed on p. 102.

The Respiratory Rhythm is often disturbed. The inverted type of rhythm, in which the pause occurs at the end of inspiration instead of expiration, is commonly seen. Most frequently it is seen in cases

of pneumonia, but it may also occur in bronchitis or even in nasal catarrh. Indeed, not a few children without any respiratory disease adopt this method of breathing when under examination, while it is also seen during sobbing.

Tachypnoea is the name given to an interesting form of increased respiration rate without any respiratory distress. It is commonly seen in children with a primary pneumonia. The respirations are quickened, the breathing being almost entirely abdominal in type, but the colour of the patient is good, and there is a total absence of any true dyspnoea. In this form of breathing the dilatation of the alveoli occurs in expiration, not in inspiration.

Dyspnoea is seen where there is obstruction to respiration, as in secondary pneumonia and many other conditions. Here the dilatation of the alveoli is during inspiration. In infants, however, movements of the nostrils are inconstant, and may be replaced by inspiratory or convulsing of the lower lip, combined with, in the worst cases, drooping

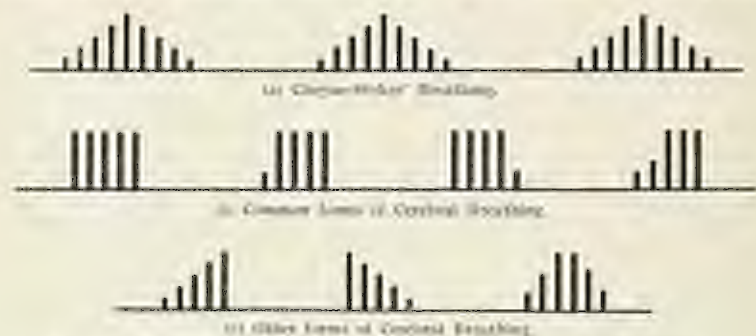


FIG. 1.—DIAGRAMS TO REPRESENT FORMS OF GROUPED OR PERIODIC BREATHING.

of the jaw or backward movement of the head during inspiration. Tachypnoea often passes into dyspnoea as a primary pneumonia turns its course. A short expiratory grunt is very common in pneumonia.

Air-hunger is seen in children in many conditions apart from diabetes. The causes of acid intoxication in children are enumerated at p. 82.

Dissociated Breathing, sometimes known as thoracic breathing, is very constant in cases of thoracic disease of any severity. It consists of the irregular alternation of thoracic, abdominal and combined respiratory movements.

Grouped Breathing is commonly seen in children. It is a sign of failure on the part of the respiratory centre. Two forms are recognized. In the first (true Cheyne-Stokes' breathing) the centre fails owing to the action of circulating toxins, as in *irremia*. It may also be seen in its slighter forms in acute diarrhoea and in pneumonia. In this type, the respiratory movements wax and wane gradually proceeding

to a maximum and gradually falling to a minimum or to temporary complete cessation. The second type of grouped breathing, known as cerebral breathing, is more common in children. In it, the respiratory centre fails as the result of increased intracranial pressure. It is seen most frequently in cases of acute meningitis, particularly in the tuberculous variety. In cerebral breathing, the commonest rhythm is that in which four or five equal or almost equal, deep breaths occur, followed by a period of apnea. Sometimes the respirations mount gradually to a maximum and then suddenly cease, or starting with deep breaths, the respirations gradually diminish. Occasionally, but rarely, a rhythm indistinguishable from that of Cheyne-Stokes' breathing is seen in cerebral breathing. The rhythm, in the latter form of grouped breathing, often changes from hour to hour.

In neither form of grouped breathing does death necessarily occur within twenty-four hours. In tuberculous meningitis cerebral breathing may last for as long as a week, and is usually present for two or three days before the fatal ending of the disease.

Yawning and sighing sometimes precede the onset of definite grouping of the breathing; such signs occurring every few minutes may give rise to a suspicion of some serious intracranial disease.

The causes and significance of cough and hemoptysis are considered in the opening paragraphs of Section VI.

The Circulatory System.—Heart.—The left border of the deep cardiac dullness is sometimes obscured by the tympanic note of the abdominal viscera. This can usually be avoided by examining the child in a sitting or standing posture. The deep dullness extends to the left to within half an inch of the left vertical nipple line, but in young children, extension to the nipple line itself cannot be regarded as abnormal. The upper limit is at the third rib. To the right, it extends to half an inch beyond the right margin of the sternum in the fourth interspace. In the third space no cardiac dullness can be detected unless the heart be dilated to the right. The apex-beat of the heart may be in the fourth space or below the fifth rib, slightly internal to and below the left nipple. Hypertrophy and dilatation of the heart produce the same changes in the character of the pulsation at the apex-beat as in adults. Precordial bulging is commonly present where there is much hypertrophy of the heart.

Chronic heart-disease in a child under the age of two years is almost invariably of congenital origin, while acquired heart-disease after this age is due, in the vast majority of cases, to a rheumatic infection.

Thrills are common in both acquired and congenital heart-disease. In the former, however, it is to be noted that presystolic thrills are unusual, while a systolic apical thrill is very frequently present in cases of mitral regurgitation with much hypertrophy of the left ventricle.

Diastolic shocks are very common in children. They are felt over the pulmonary area in many cases of rheumatic dilatation of the heart.

A pulmonary systolic bruit is often present in a similar condition. It is probably due to a dilated pulmonary cone.

On auscultation, particular attention should be paid to the second cardiac sound just internal to the apex beat of the heart. Localized to this area, the "pseudo-reduplication" of the sound and the mid-diastolic murmur are first heard. The significance of these signs is discussed under Mitral Stenosis.

The Genito-Urinary System.—In the male the examination of the genital organs should not be omitted. The occurrence of tuberculosis epididymitis or of syphilitic fibrosis of the testis may be of considerable aid in diagnosis.

The normal and abnormal conditions of the urine are dealt with at the beginning of Section VIII.

The Blood.—The blood in infancy and childhood is described in Section IX.

The Nervous System.—The most difficult but at the same time the most interesting problems in children's diseases occur in connection with nervous diseases and symptoms.

In the first place, it must be remembered that the co-ordination of the various parts of the brain is less perfect in children than in adults. More particularly is this so in the case of infants. In this connection may be mentioned the important fact that, in an infant, a unilateral convulsion does not necessarily mean any local damage to one side of the brain. Owing to the considerable lack of co-ordination between the two halves of the brain in an infant, a unilateral convulsion may result from some reflex cause, such as gastro-enteritis, that might be expected to produce a generalized convulsion.

Again, in children the disproportion between functional disturbance and organic disease is particularly marked. Very commonly there is such an exaggeration of a symptom which is due to an organic lesion, that there is great danger of regarding the whole condition as functional in type. A child, for instance, with a spastic weakness of an arm due to an intracranial tumour, may complain of total inability to move the limb. Although, when the attention is distracted by conversation, it is seen that the arm can be held out straight and moved with fair power. In such cases, therefore, as appear to be functional, the greatest care must be taken to exclude some co-existing organic factor in the production of the symptoms. Not seldom a functional paralysis is due to a past condition; for instance, rheumatic pain in a joint may lead to immobility of the limb outlasting the period of pain.

Conversely, the functional derangement may be very much less than the extent of organic disease might be expected to produce. This is particularly well seen in the case of tuberculous tumours of the brain.

Nervous signs may be given in a condition entirely separate from

the nervous system. A functional paralysis may be set up by some painful state that is past, as has been mentioned. A pseudo-paralysis may be due to some painful condition, such as subperiosteal abscess or hæmorrhage, which causes the child to keep the limb immobile. The pseudo-meningeal condition, spoken of as meningismus, which occurs in many conditions in children apart from meningitis, is an example of nervous symptoms arising from a disease not directly connected with the nervous system.

Mental Condition.—The signs of normal mental development are given on p. 22.

A mentally backward child, as Dr. West has said, is one who would be normal at a younger age, while a mentally deficient child would be abnormal at any age.

The memory may be tested by asking the child to repeat various nursery rhymes, and other tests may be applied in arithmetic or geography.

The child's position in school is not an accurate measure of its mental development in hospital patients. Formerly, the child entered the first standard when seven years old, and if normal passed into a higher standard each year, until reaching the seventh at the age of thirteen. This system of standards is, however, rapidly passing away, and the children are grouped in different classes for different subjects. A comparison of the age of the other children in the patient's classes is of no value, for in most schools backward children are promoted so that they do not mix with those very much younger than themselves. Care must also be taken in judging of a child's mental development by asking it questions about its school work, for education is nowadays directed towards teaching the child to observe rather than to acquire facts. It learns, for instance, the sounds and not the names of letters. Multiplication tables, facts in geography, dates in history, are not taught as such, but lessons are given in which various elementary facts occur.

The Motor-power of the limbs in older children may be tested in the usual way: in infants it is only necessary to watch the movements which occur. A paralysis may be of congenital origin, in which case it may not be observed until such time as the child should begin to walk. It may come on gradually, as in post-diphtheritic neuritis, or suddenly, as in acute poliomyelitis. It may be due to some painful condition such as syphilitic epiphysitis, scurvy, or osteomyelitis, in which case it is known as a pseudo-paralysis. A functional paralysis may result from some past painful condition of a limb, or may be grafted upon a weakness of organic origin. Lastly, simple weakness of the limbs may prevent their being put to their proper use, as is so commonly seen in rickets, in which the child "goes off its legs," although it can kick them about freely.

The Cranial Nerves may for the most part be easily tested. Movements of the eyes may be induced by attracting the child's attention

by means of some bright object, or by a noise. Movements of the lips may be brought out by getting the child to blow out a lighted match if it refuse to try to whistle. Sometimes the first sign of facial weakness is seen in the occasional absence or diminution of the natural blinking movement in one eye. Retraction of the upper eyelid is a very important sign of posterior basic meningitis. It is also seen, although less constantly, in other cases of hydrocephalus (Fig. 42).

Hearing is tested with difficulty. Loud and unexpected noises may be used, but it is difficult to be sure that the child does not see the source of the production of the sound. The best test in the most difficult cases is to get the child's mother to call it by name from another room. The possibility of congenital deafness must be remembered.

The sight is best tested in an infant by moving a bright light about in front of its eyes, observing if the child takes notice of it or fails to follow it with its eyes. The feeding-bottle may be used in the same way. Threatening the cornea with the finger is not a satisfactory method of testing the sight in infants. The loss of sight in an infant usually signifies posterior basic meningitis.

Ophthalmoscopic Examination.—An examination by the direct method is usually quite possible in children, if the pupils have been artificially dilated. The child may be placed on its side at the edge of the bed or lying on its back looking upwards. Older children, and those in good health, may be best examined while sitting up. Whatever position is adopted, the important point to be observed is to avoid obscuring with the head the view of both of the child's eyes. One eye must be left so that the child's attention may be fixed upon some object.

It is important to remember that in infants the optic disc is normally very white and clear cut, suggesting atrophic changes. The vessels, however, are of good size. In older children, the disc is perhaps rather more pink than in adults.

Optic neuritis is extremely rare in infants, owing probably to the fact that in them intracranial pressure is relieved by the bulging of the fontanelle and widening of the sinuses.

Chorioid telangiectas appear as small whitish grey areas in the peripheral portions of the field, normally in close proximity to a blood-vessel. Their presence signifies acute primary tuberculous of the choroid, but they are very rarely present sufficiently early to be of use in diagnosis. They have to be distinguished from areas of exudate or of chorioid-retinitis.

The Ears are examined with difficulty in a child. The external auditory meatus, it must be remembered, is short, and the membrane obliquely set. Otitis media may be present without any otoscopic signs.

The Larynx can only be viewed by some type of direct laryngoscope used under an anæsthetic. By this means, however, a good view can be obtained.

Reflexes.—These are usually easily elicited. The necessary muscular relaxation of the limbs is most successfully obtained by engaging the child in conversation. For the knee-jerk, the ordinary method of reinforcement is of no use save in the oldest children; for straining of the arms as a rule leads to straining and stiffness of the legs. The commonest cause of loss of knee-jerks in a child is post-diphtheritic neuritis.

The other tendon reflexes may be obtained in the usual ways. Those of the arms are rather difficult to elicit unless they are brisk. Ankle clonus should be tested for by dorsiflexing the foot very gently while the knee is flexed. The abdominal reflexes in children are brisk, but disappear quickly in many toxic conditions associated with drowsiness or unconsciousness, as well as in organic lesions of the pyramidal tracts.

The *plantar response* up to the age of two years is of the infantile type, consisting of extension of the big toe with a spreading movement of the others. After this age it gradually alters to the adult flexor type. It is not uncommon, however, for any illness which produces cerebral symptoms to bring back the infantile type of response in children up to about six years of age. A true extensor response, a deliberate extension of the big toe and slight flexion of the little toes, is of more value in diagnosis.

Kernig's Sign.—This is tested in the following way. The child is placed upon its back. The examiner, with one hand on its knee and the other under the heel, bends the lower limb until the thigh is at right angles to the trunk and the leg at right angles to the thigh, and then attempts by elevating the foot to straighten the leg at the knee. The sign is positive if the rigidity of the limb is too great to allow the straight position to be attained. At an earlier stage, before actual limitation of movement develops, pain may be caused by an attempt to elicit the sign, and this should be regarded as suggestive of concurring rigidity.

Kernig's sign is thus merely an index of the spasticity of the lower limbs. It is of course an important sign of acute meningitis, especially of the tuberculous variety, but it is to be remembered that it may be positive in all conditions in which meningismus is present. Probably the commonest cause of a positive Kernig's sign is acute primary pneumonia. It is frequently present in rotic fever.

Rigidity of the upper limbs may easily be tested by taking hold of the wrists and repeatedly flexing the arms. Rigidity of the neck may be appreciated by attempting to raise the child's head from the pillow, or by eliciting it, if conscious, to follow objects with its eyes and head.

Lumbar Puncture.—This has become a most important method in diagnosis. In children it is very easily done, often without an anæsthetic. It produces no bad effects unless more than one ounce of fluid is withdrawn.

Discharge	Appearance	Cells	Organisms, etc.
Purulent meningitis (active stage)	Turbid	Mainly polymorphs	Meningococci (few)
Purulent meningitis (late stage)	Clear	Mainly lymphocytes	Meningococci (few)
Pneumococcal meningitis	Turbid	Mainly polymorphs	Pneumococci
Tuberculous meningitis	Clear or opalescent	Mainly lymphocytes	Tubercle bacilli
Tuberculous tenosyn- ovitis (in inguinal)	Clear	Some excess of lymphocytes	No tubercle bacilli
Intraocular syphilis	Clear	Some excess of lymphocytes	Wassermann test
Intraocular abscess	Clear	No excess of cells	Nil
Acute polymorphitis and osteophalitis	Clear	Slight excess of lymphocytes	Nil
Chorea	Clear	Usually no excess of cells	Sometimes diplococci
Dactylitis	Clear	Distributed number of cells	Nil
Erysip	Discoloured	Red blood corpuscles	Nil

Table 2.—Characteristic Features of Various Types of Inflammation in Connective Tissue

Method.—The child is conveniently put on its right side, the trunk flexed as far as possible (an important point) and its limbs wrapped up in blankets. A medium-sized needle, such as is used for exploring the chest, and at least $\frac{1}{2}$ inches in length, is sterilized. A line is drawn between the highest points of the iliac crests posteriorly—the passes over the tip of the fourth lumbar spine. The skin here is carefully sterilized. The needle is then inserted half an inch above or half an inch below the fourth spine. In children the puncture is most conveniently made exactly in the middle line, not to one side. The needle is passed in a slightly upward direction to the depth of $\frac{1}{2}$ inches. A common error is to fail to go sufficiently deep. The fluid should be collected in sterilized test-tubes. It should be noticed if it runs (i.e., is under pressure), or only drips, as is normal. If the first fluid that comes away be blood-stained, this should be collected, as it is of value for a bacteriological examination; but as soon as it runs clear, another specimen should be collected in a second tube, for examination of the cells in the fluid. Occasionally in suppurative meningitis, the exudate is too dense to pass through the needle. Suction should not be applied where no fluid is obtained. On withdrawing the needle the opening in the skin is sealed with a collodion dressing.

The therapeutic uses of lumbar puncture are few. It will relieve convulsions in meningitis, and the symptoms in some cases of meningismus. Severe motor chorea is temporarily relieved by it.

Diagnostic Value.—The chief use of lumbar puncture is as an aid to diagnosis, and here it is quite invaluable. The points in the examination of the fluid of diagnostic value in various diseases are given in the preceding table (Table 2).

SECTION II, THE DEVELOPMENT AND FEEDING OF CHILDREN.

I—PHYSICAL AND MENTAL DEVELOPMENT.

In order that we may appreciate the more readily any departure from the normal, we must be familiar with the ordinary course of development in infancy and childhood. More especially is this of importance in estimating the progress of mental growth, for during infancy backwardness in this direction has to be recognized by what the child fails to do rather than by what it does.

While it is impossible to burden the mind with many dates in this connection, the most important of them quite sufficient for all ordinary purposes, can be retained in the memory with ease. All the figures are, however, merely approximate, being founded upon averages. They must not therefore be regarded with too much reverence, for within the limits of health, both physical and mental, there is room for wide variations from the averages given.

PHYSICAL DEVELOPMENT.

Weight.—The average weight of an infant at birth is between 7 and 8 lb. During the first few days it loses weight to the extent of 6 or 8 oz. After this, however, it begins to gain at the rate of about 5 oz. a week ("on once a day and two on Sundays")—although considerably less than this (4 to 6 oz.) is quite satisfactory progress. In quite healthy infants, the gain in weight is often rather irregular. By the fifth month, the birth-weight should be doubled, at the twelfth month trebled, and at two years quadrupled. From this time onwards the weight increases at the rate of four to six pounds a year, the older the child gets the quicker being the progress.

The most convenient numbers to commit to memory in this connection are the multiples of the birth-weight, taken as seven pounds.

At birth	7 lb.
At 5 months	14 lb.
At 12 months	21 lb.
At 2 years	28 lb.
At 7 years	49 lb.

Weight-Chart.—It is often convenient to use a weight-chart to record the progress of a case. Such a chart should be applicable to patients of all ages, should contain no record of "normal" weights (which often unnecessarily worries the parents or patient), and should be capable of registering half-ounces. In order to meet these wants I have devised

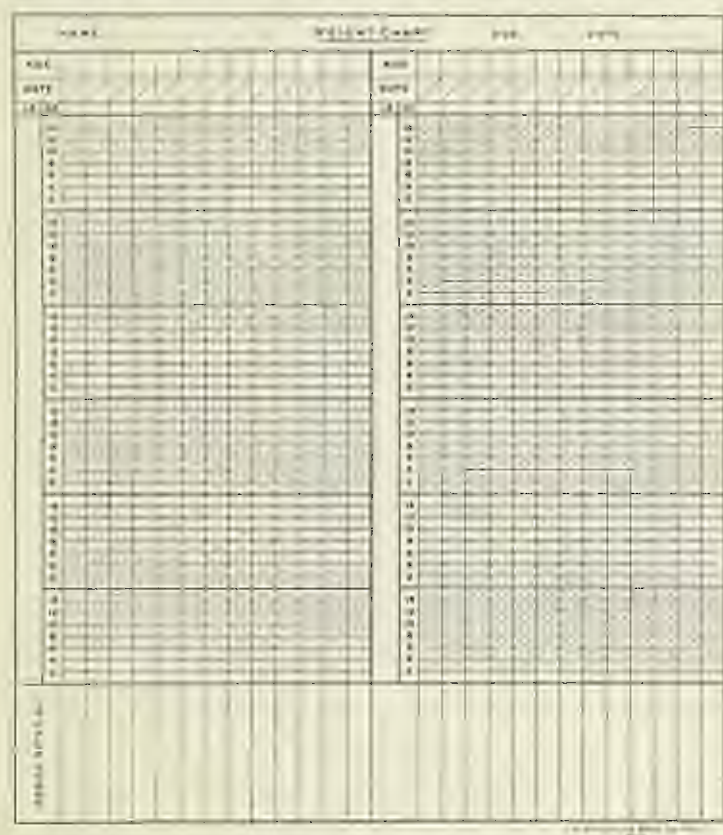


Fig. 4.—WEIGHT CHART (9 SCALES).

the chart which is illustrated here, and which is printed by the publishers of this book. The chart in use is shown on page 49.

Normal infants should be weighed once a week. Delicate infants may be weighed twice a week, but not, as a rule, more often.

Length.—At birth, the length of an infant is about 19 in. During the first year it grows at the rate of about $\frac{1}{2}$ -in. every month, and at

twelve months its length is 27 in. After this, until the age of five years, it grows about $\frac{1}{4}$ in. in each year, and from five years old until puberty at the rate of about 2 in.

Size of Skull.—It may be of some moment to know the average measurement of the circumference of the head. The maximum circumference is most conveniently taken—

At birth	15 in.
At 6 months	16 in.
At 12 months	18 in.
At 3 years	19 in.
At 6 years	20 in.
At 12 years	24 in.

The Fontanelles.—The anterior fontanelle should not measure more than an inch in length or breadth at the twelfth month. It should be closed by the eighteenth month certainly before the end of the second year.

The lateral fontanelles are usually closed at birth, and the posterior fontanelle by the second month of life.

Dentition.—The eruption of the first tooth varies between the fourth and eighth months, and usually occurs about the sixth month. The lower central incisors are the first to appear; they are followed by the upper central incisors and the lateral incisors, first in the upper and then in the lower jaw. At twelve months 8 teeth should be present, and the milk-dentition (20 teeth) should be complete by the second year.

Milk Dentition		Permanent Dentition	
	Months		Years
Central incisor	8	Central incisor	7
Lateral incisor	7	Lateral incisor	8
Canine	15	Canine	11
1st deciduous molar	12	1st bicuspal	9
2nd deciduous molar	24	2nd bicuspal	10
		1st molar	6
		2nd molar	12
		3rd molar	24

Table 1.—TABLE OF ERUPTION OF TEETH.

Occasionally a tooth is present at birth, but is seldom well developed or well placed. Dentition tends to be late in its appearance in rickets; or having started, it may cease during the active stage of the disease. In inherited syphilis, teething is said to start early, but this is doubtful.

The second dentition starts with the eruption of the first molar (the six-year-old molar) at the sixth year. It consists of 32 teeth, of which all except the wisdom teeth are set by the twelfth year.

The dates of eruption of the teeth are best remembered by considering the teeth in order, starting from the middle line of the jaw. This leaves only one set of points to be remembered. In the permanent dentition it may be noted that the figures for the five first teeth run in order, except that 14 is interpolated in the middle, while the dates of eruption of the last three teeth considered are multiples of each other.

Pulse-rate.—At birth, the pulse-rate is about 130 per minute. By the end of six months it drops to about 110, and is more regular in rhythm. The respiration-rate at the same ages is about 30 and 20 per minute.

Stools.—During the first two months the motions number four a day, and after the first week are yellow, and of a slightly sour, non-feculent odour. Gradually they become darker in colour, more feculent, and less frequent. During the second year the bowels act twice in the twenty-four hours, and during the third year the motions become properly formed.

Movements.—These are described under mental development, upon which to a large extent their appearance depends.

The characteristics of the blood and urine and other matters are described in the introductions to articles dealing with the systems in question.

MENTAL DEVELOPMENT.*

At birth the infant will suck but will not seek the breast. A bright light causes blinking of the eyes within twenty-four hours of birth, while tactile sensation and taste seem present from the beginning. Hearing is probably absent at birth, but shrill sounds appear to be recognized within two or three days of birth. By the fourth week the sense of hearing is good.

At birth the movement of the eyes is quite inco-ordinate, but by the sixth week this becomes normal, convergence for near objects is seen, and the child will sometimes follow bright objects with its eyes. About this time it will turn its eyes, or peck out, towards objects, but as yet there is no space-perception, so that it will perhaps reach for the moon. Its movements gradually become more purposeful and less aimless.

At the beginning of the third month the infant turns its head to follow objects with its eyes. By the fourth month, if it is set up, it can hold its head still and erect. By the sixth month it carries things to its mouth and grasps all objects firmly, but does not for another

* Most of the facts and dates in this article are taken from Dr. W. H. R. Stoddard's paper "On Instinct" (*Journal of Mental Science*, July, 1886).

couple of months acquire the idea of letting things go again. At the eighth month it takes pleasure in making a noise.

At nine months the child should be able to sit up without support. At ten months it begins to stand and at twelve months can walk with only slight assistance. By eighteen months it should walk well.

The smile appears at about the sixth week, and laughter at the end of the fourth month.

During the third month the infant begins to imitate sounds. During the ninth month the first words are spoken. These are imitative and not voluntary, and consist of the sounds "kak-kak," "da-da," etc.

Gradually, voluntary language begins to appear and is learnt by imitative imitation. It attempts to say "yes" and learns to say "ya" when given anything. It will not at first say "ya" when told to do so, for that would be a volitional act. At first "da-da" stands for any man, not necessarily for the child's father. "ba-ba" for anything soft and furry. About the fourteenth month these words are used only for their proper objects, and several other words are added to the child's vocabulary. At eighteen months small sentences of the "go la-da" order are made, and by the end of two years the child's talking should be fairly good.

Habits of cleanliness, if the child has been carefully trained, should be acquired by the eighteenth month, although nocturnal incontinence may continue until the second or third year unless the child is purposely awakened at night.

Various other instincts continue to develop. Curiosity appears at about the eighteenth month. A little later the child begins to lose its dislike of strangers which it has shown since about the fifth month. During the third year the instinct of make-believe and some idea of time appear. Destructiveness and disinterested cruelty develop about the fifth year; constructiveness two years later. Rivalry the collecting instinct and greediness become prominent during the last years of childhood, from the ages of seven to ten.

In sketching the mental development of a child many dates have been mentioned which are of little interest from a strictly medical point of view. It will be well, therefore, to give such points in one usefully remembered in a table form—

	Normal Development	Severely Delayed
Holding up head	4 months	6 months
Sitting up	9 "	12 "
Walking	12 "	25 "
Talking	12 "	18 "
Chattering	18 "	24 "

TABLE I.—NORMAL COURSE OF THE MENTAL DEVELOPMENT FROM CONCEPTION.

The figures given in the second column represent the latest ages by

which the various developments should have appeared in a child who is physically and mentally normal. Where the physical development is good, delay in appearance of the faculties named beyond these dates should suggest mental underdevelopment. They are, however, variable within the limits of health, particularly is this the case in the development of the power of talking.

II.—THE FEEDING OF HEALTHY CHILDREN.

BREAST-FEEDING.

Up to the age of nine months the infant is best fed on its mother's milk. By this means it receives a milk which is sterile, which contains human proteins, and which forms a scarcely perceptible clot when it is acted upon by the gastric juice. Every effort should therefore be made to secure for the child its most nourishing food.

It has been shown many times that the death-rate amongst artificially-fed infants is very much higher than in the breast-fed. In not a few weakly infants, human milk is the only food that can be well assimilated. During the first few weeks of life breast-feeding is of special value, so that if the infant has to be weaned, some of the most dangerous weeks, at all events, will have been satisfactorily got over.

Human Milk.—We have to consider here the composition, quantity, and chief abnormalities of breast milk.

Composition.—Colostrum, the fluid secreted by the breast for the first few days after parturition, is richer in soluble proteins, but contains considerably less fat than does the later milk.

When lactation is fully established, the milk varies in its composition according to whether it is the early or late milk to leave the breast. The first portion is the most watery, and the last portion the richest milk. For analytical purposes, therefore, a sample must be taken from the whole contents of the breast or, more conveniently, from the middle third of the milk obtained by emptying the breast.

The percentage composition of human milk is given and compared with that of cow's milk in the following table:—

	HUMAN MILK		COW'S MILK	
Protein	Casein Lactalbumin	% ± per cent	1.25 75	4.0 per cent
Fat	—	3.3	—	3.3
Lactose	—	2.8	—	4.0
Salts	—	.2	—	.7
Water	—	87.1	—	87.8

TABLE 5.—COMPARISON OF COMPOSITIONS OF HUMAN AND COW'S MILK.

Of these figures, the percentages of the protein and fat have at least to be remembered. While in human and cow's milk fat is present in equal proportions, there are marked differences in the protein-content, and particularly in the proportion of the casein-forming protein caseinogen (calculated as casein).

The specific gravity of human milk is about 1030, and its reaction neutral or faintly alkaline.

Quantity.—The amount of milk secreted daily varies from half a pint during the first weeks to two pints during the last months of lactation.

Analysis.—Where it is necessary to make an analysis of the milk, a sample of the middle third of the flow should be examined. This is easily obtained by putting the infant to the breast for just one-third of the time that is usually taken to empty it, and then drawing off some of the next milk by a breast-pump or by squeezing. For our purpose an ounce of milk is a convenient quantity.

The specific gravity, which can be easily taken by means of a small areometer, is no guide to the composition of the milk, unless the percentage of fat or of protein is known. Fat being lighter than water,



FIG. 53.—TYPE FOR USE IN CENTRIFUGE FOR DETERMINING THE PERCENTAGE OF FAT IN MILK. (BARD & TATLOCK.)

and protein being heavier, a high specific gravity with a high percentage of fat indicates excess of proteins, while a low specific gravity with a low percentage of fat points to a deficiency of proteins.

There are now many laboratories from which a complete analysis of a sample of milk can be obtained. For ordinary purposes, the proteins being estimated with difficulty, the amount of fat is calculated.

Determination of Fat.—This can easily be done in a centrifuging apparatus, small tubes being specially made for the purpose. Such a tube, made by Messrs. Bard & Tatlock is shown here (Fig. 53). For it less than half an ounce of milk is required. If a centrifuge cannot be used, recourse may be had to a tube of equal calibre throughout, which is graduated empirically in percentages of fat. The milk is placed in a tube, corked, allowed to stand for twenty-four hours, and the amount of fat read off. This gravity method is however very inaccurate.

Determination of Protein.—The amount of protein can be calculated with sufficient accuracy by the consideration of the specific gravity of the milk where the fat-percentage is known. By this means, excess or deficiency of protein can be judged, as has been mentioned above.

Microscopical Examination.—This is of no use in determining the fat-content of milk. Colostrum corpuscles should disappear from the

milk in from one to two weeks of the beginning of lactation. Pus, blood, and micro-organisms can be detected by this means.

Management of Breast-Feeding.—We have here to consider the feeding of the child until it is weaned, the hygiene of the mother and of the infant, and the various abnormalities which may occur in breast milk.

Feeding.—During the first day the infant should be put to the breast every six hours, during the second every four hours, and on the third day, when the flow of milk is established, the regular feeding ten times in the day—two hourly by day and four hourly by night—should be started. This is continued until the end of the second month.

Before the flow of milk is established the child is usually satisfied by the colostrum which it obtains from the breast. Thirst seems to be more marked than hunger at this age. A little water may therefore be given. Should the infant be poorly developed, or should there be excessive loss of weight, accompanied by some rise of temperature (the so-called *marasmus fever*), a 2 per cent solution of lactose or some whey, may be administered.

From the beginning the child should be fed regularly, and after each feed should be laid down. It should be roused if necessary when the next feed is due. On no account should the mother give extra feeds to quiet it.

Crying does not always indicate hunger. It may be due to thirst, cold feet, cold, wet napkins, pneumonia or hyperacidity of the stomach. Nothing is more likely to give rise to future trouble than the habit of putting the child to the breast whenever it cries. Nor should it be taken up in the arms directly it cries: such indulgences end, sooner or later, in the child becoming a burden to the household.

Nevertheless, crying may be a sign that there is something wrong in the breast-milk. When the infant sucks vigorously but very soon fruits and cries, the breast probably contains little or no milk for it. If, when taken from the breast, the child cries for a time, then sleeps, but wakes before the next feed is due, it is evident that it has had an insufficient amount of milk. Where the flow of milk is too copious, being often of poor quality, the child falls asleep directly after it is fed, so the normal infant should, but soon wakes again, crying with colic.

In such ways as these the infant gives us some indication as to the amount of milk which the mother is able to give it, and these are much more satisfactory guides than the appearance of the breasts, for large breasts do not always give the largest quantities of milk and small breasts may yield quite a sufficient amount. The only sure method of ascertaining the amount of milk obtained at each feed is to weigh the child before and after it has been put to the breast. For this, however, delicate scales are necessary.

The various faults of breast milk and the measures by which they can be corrected, are mentioned later (p. 28).

After the end of the second month the infant resolves its stools at longer intervals. At six months it can usually pass the night of eight hours without nourishment.

It must be borne in mind that the infant's progress as estimated by its gained weight will necessitate changes in whatever scheme may be drawn up, and in the following table the numbers are only approximate.

Age	NO. OF FEEDS IN 24 HOURS	INTERVAL (IN HRS.)	FEEDS WHEN NIGHT SLEEPS
1st day	4	6-hourly	1
2nd day	6	4-hourly	2
3rd day to 3rd month	10	3-hourly	2
3rd month	8	2½-hourly	1
4th and 5th months	7	3-hourly	1
6th month	6	3-hourly	0

Table 4.—Schedule for breast-feeding.

The amount of each feed is to be judged by the infant's appetite. Usually from ten to fifteen minutes at the breast should be given, but the child should not be encouraged to take more than it wants.

The rapidity with which the milk is taken is also of importance. If the flow is too free, it is liable to set up vomiting and colic, and the mother should be instructed to regulate it by compressing the nipple gently between the first and second fingers as she supports the breast in the palm of the hand in the usual way.

Hygiene of the Mother.—For some weeks before the baby is born, the breasts may well be given attention. The nipples may be lathered in spirit and anointed with liniment. In many women, however, such measures are quite unnecessary. When nursing has started, the nipples should be washed with sterilized water before and after each feed and carefully dried.

Throughout the lactation period, the mother's food must be plentiful, the amounts of fluid and solid food being sufficient to allow of a full secretion of milk without draining the woman's resources.

On the first day fluid food only should be given. It must consist chiefly of milk and gruel. Broths may also be given. A cup of tea is usually extremely well received and there is no adequate reason why it should be withheld. On the second day much the same diet is required. To it may be added cream-thick or light milk-pudding. Thin slices of bread and butter may be given. On the third day a more normal diet may be allowed should the patient's condition be satisfactory.

During the nursing period, plenty of milk, at least a quart daily, should be taken by the mother. The food should be plain: eggs,

meat, cereals and fruit. Green vegetables should be taken only in moderation, as in some cases they seem to upset the child at the breast. Tea and coffee should be taken only in small quantities; alcohol is best avoided. Cocoa forms a very useful beverage.

As soon as the mother is allowed out of doors, driving exercise should be taken. Later, walking should be begun, and throughout the nursing period some exercise of this kind should be the rule. Actual fatigue, however, must be avoided. There is no harm in allowing a healthy infant occasional feeds from a bottle, in order that the mother shall not be wearied by the office of nursing.

After child-birth most women are anæmic, and this condition should be treated by iron. Often by this means the secretion of milk is greatly improved. Constipation may be treated by means of cascara. Hydragogue cathartics and saline aperients are contraindicated. Sulphur, rhubarb, scenna, jalap, scammony, saline aperients and castor oil, may be excreted in the milk.

Other drugs which are excreted in the milk are opium, iron, potassium iodide and bromide, mercury, arsenic, belladonna, and volatile oils. Alcohol may cause indigestion in the child when taken by the mother, especially if in any considerable amount.

Menstruation occasionally causes the milk to disagree with the infant. In such a case, hand-feeding should be substituted for a few days, the breasts being emptied periodically by a breast pump.

Nervous and emotional influences are very prone to cause some change in the quality of the milk. For this reason the nursing-mother, while allowed plenty of time for recreation and exercise, should not be permitted much in the way of excitement. Outbursts of passion or of grief cannot always be avoided.

Contraindications to Nursing.—Apart from the causes already mentioned, which may necessitate temporary artificial feeding, there are four absolute contraindications to breast-feeding. One of these is pregnancy. As long, however, as the infant continues to increase in weight, nursing during the early days of pregnancy does no harm, and thus it may be possible to postpone weaning until a satisfactory time, as, for instance, prevalence of cooler weather. Tuberculosis, chronic nephritis, and mental disease, all contraindicate breast-feeding for the sake of the mother or child.

In the case of supposition in one breast, in the absence of much constitutional disturbance, the infant should be fed from the other, supplemented if necessary by hand-feeding.

Hygiene of the Infant.—When the baby is born it is wrapped in warm blankets; its eyes are bathed in a solution of perchloride of mercury (1-10,000), followed by weak boracic lotion. As soon as circumstances permit, the child is given its first bath. Having been wiped over with a clean soft cloth, it is washed with soap in a bath at 66° in front of a fire. When thoroughly dried, it is powdered all

over with toilet powder. The cord is covered with an aseptic soft dressing and well powdered with boracic powder. This is kept in place by a flannel binder six inches broad, the end of which is sewn. The child is then clothed in flannel.

The method of feeding has already been detailed. After each feed the infant should be kept as still as possible. Nipples should be changed before rather than after a feed. Care should be taken with feeble children, or those that are born much exhausted, that good crying efforts are encouraged.

After each feed the nurse should cleanse the mouth and gums by gently spreading them over with cotton-wool moistened in clean water.

Between the second and third months in summer and the third and fourth months in winter, the child is "short-coated," and the doctor must begin to be on his guard against the vagaries of the "cobbling" and "hardening" parents alike.

Abnormalities of Breast Milk.—Mention has already been made of the ways in which the infant indicates that the milk is deficient in quantity, or, while usually copious, is poor in quality. The condition of the digestion, the character of the stools, and the general progress, will also show whether the milk is satisfactory or not, and to some extent what is wrong with it. The methods of gauging the amount of milk taken in each feed, and of estimating the quantities of its constituents, have also been described (pp. 24-5).

The milk may be deficient in quantity or quality as the result of maternal worry, anemia, or constipation. Not seldom the mother is upset because she thinks her milk is unsatisfactory, and then in its turn tends to make it worse. She should therefore be reassured on the point. In many cases, the administration of iron produces a rapid improvement in the milk. With the mother in apparently good health, the constituents or quantity of the milk may still be at fault.

Deficiency in Quantity.—Rest for a day or two in bed or on a sofa often has a beneficial action in promoting an increase in the flow of milk. Extra milk, greek, and cocoa may be added to the diet. Alcohol, given *ad libitum*, is not to be recommended, for, amongst other reasons, it often upsets the baby. Malt extract will often promote an increased secretion of milk, although its chief action is in increasing the amount of fat. Deficiency in quantity and in quality usually occur together.

Deficiency in Fat.—The amount of fat in the milk may be increased by ordering a diet richer in proteins and fats. Meat in all its forms, poultry, fish, and eggs should be taken in increased amounts. Malt extract is of value.

Deficiency in Proteins.—Here the same principles hold good. The exercise should be diminished and the diet increased.

Excess of Proteins or Fat may be treated by ordering more exercise and diminishing the diet, the former reducing particularly the proteins, and the latter the fat.

Where, in spite of treatment, the milk remains too rich, we may circumvent the difficulty in various ways. The infant may be given only the "fore-milk" (that secreted first), sucking for five minutes at each breast at every feed. Or we may give the child a teaspoonful of water immediately before it is put to the breast, in order to dilute the feed. In the same way, two grains of sodium citrate, or two grains of sodium bicarbonate may be given in a drachm of water in order to render the clot less tenacious and to promote digestion.

WEANING.

Where the choice of the time for weaning is in our hands, the end of the ninth month is to be preferred. A time should be chosen when the child's digestion is in good order. Weaning during a spell of hot weather is to be avoided whenever possible. It is better to let the infant's weight remain stationary for a time, until the temperature of the atmosphere is lowered.

Weaning should be done gradually; and this is perhaps the most important point. In many cases, for several weeks perhaps, the infant has been accustomed to a feed from the bottle once or twice in the twenty-four hours, and here there is seldom much difficulty in getting the child off the breast altogether. Where it has been entirely breast-fed, the bottle should be given once a day, then twice, until at the end of a fortnight, or in delicate children at the end perhaps of a month, it is completely weaned.

Occasionally the child resolutely refuses to start bottle-feeding, and here it may be necessary to wean it abruptly. If no food be given for several hours the infant in a rare quickly comes to terms.

WET-NURSING.

Wet-nursing is comparatively seldom adopted in this country. It may be difficult to find a wet-nurse; or her milk may not suit the child. In other ways, particularly in domestic matters, the wet-nurse may not be a success. On the other hand, in some difficult cases such a method of feeding may be quite invaluable to the infant.

The wet-nurse's baby should be of the same age as the foster-child. The best criterion of the satisfactory condition of her milk is the progress which her own infant has shown. The health, cleanliness, and habits of the wet-nurse have to be enquired into, and her baby should be examined for any evidence of disease.

In many cases where the foster-child is delicate, the wet-nurse's milk will keep up its best standard if she is allowed to nurse her own infant partially or wholly as well. Needless to say, under no circumstances is it permissible for a syphilitic infant to be suckled by a healthy wet-nurse.

ARTIFICIAL FEEDING.

In the artificial feeding of healthy infants, which we are here considering, we have only to deal with the use of one nutrient food, namely cow's milk. Other preparations suitable for the feeding of children when cow's milk is unsatisfactory, are considered later.

The Bottle.—A cylindrical bottle with a flat bottom and a neck wide enough for purposes of cleansing is very satisfactory, and is particularly useful where sterilization of several feeds, each in its own bottle, is practised. In a cylindrical bottle no air-inlet is present, so that chewing movements of the jaw are encouraged as in breast-feeding. A hour-shaped bottle provided with an air-inlet may be used. An eight-ounce bottle is the usual size, but at first it may be convenient to make use of one of smaller capacity. The markings on



Fig. 1.—A pear-shaped bottle (note absence of screws and sharp angles).

the bottle, intended as measures of volume, should be tested, as they are frequently very inaccurate.

Whatever shape is employed, no undesirable tubing is permissible. The bottle should be of such a sort that the teat can be slipped over the mouth without any further adjustment being necessary, and should be devoid of screws or sharp angles.

During use, the bottle may be surrounded by warm flannel: but no cozy should be permitted for keeping the food warm for any considerable time before use. After the feed is over, the bottle should be rinsed out with cold water, followed by very hot water, and then inverted in order to dry. Before use, the bottle is washed out with hot water, or preferably boiled.

The Teat.—Sufficient attention is not always paid to the teat. It should be made so that it can be turned inside out for purposes of cleansing. It should be tested before use. It should not be too large, nor too long. Its aperture should be circular and should allow the milk, when the bottle is inverted, to escape at the rate of about a drop per second. Attention to this last point may be of considerable importance. In the case of teats that permit the milk to flow through in a stream, indigestion is very likely to be set up, owing to the rapidity of the feeding. On the other hand, a feeble infant should the teat be too hard and its hole too small, may become tired

out and leave off sucking before it has had sufficient milk. Where a cylindrical bottle with no air-inlet is used the teat must not be too easily collapsible.

The use of a "confederator," that unauthorized ornament of babies, should be discouraged as far as possible.

The Milk.—The milk should be obtained from some clean dairy which is subjected to periodic inspection. It should be conveyed to the house as quickly as possible, and delivered in air-tight bottles. Unfortunately, however, it is necessary to add that its reception in an air-tight bottle is not always a guarantee that it has not been put therein a moment previously in the road, in close proximity, it may be, to a dust-cart. No preservatives should be added to the milk.

Milk which is sold as obtained "from one cow" is not to be recommended. If truly from one animal alone, it is much more liable to vary in its composition from day to day than is the mixed milk from many cows.

Pasteurization, Sterilization, and Scalding.—It is certainly unsafe to give raw milk, especially in towns and during the hot months of the year. The presence of tubercle bacilli in milk has been demonstrated to be far from rare, while the organisms causing acute diarrhoea, although their nature is unsettled, seem to be definitely associated with a contaminated milk supply. For these reasons, therefore, we advise conscientious feeding on raw milk.

It must be our endeavour to give the child not a milk which has been sterile, but one which is so at the actual time of ingestion. For this reason, the sterilization of milk in a series of bottles, each one of which contains a separate feed, is the plan that should be adopted wherever practicable. As has been shown elsewhere (p. 74), the administration of the whole content of a bottle of sterilized milk seems to do away with all danger of scurvy which may arise by feeding from the upper layers of a quantity of milk which has been sterilized.

Pasteurization.—In this method the milk is heated to 155°-170° F. (75° C.) for twenty minutes. By this most of the common organisms found in milk, such as those of tuberculosis, diphtheria, and typhoid fever, and the streptococcus, staphylococcus and *B. coli* communis are killed; but the spore-bearing organisms are not destroyed. Pasteurization does not alter the taste of the milk, nor does it interfere with its digestibility. The chemical constitution of the milk is not much altered, or at least not sufficiently to add much to the likelihood of the production of scurvy.

Where possible, pasteurization should be carried out in an apparatus made for the purpose, in which there is a rack to hold the bottles required for twelve hours' feeding (fig. 7). The mouths of the bottles may be sealed by sterilized wool, or preferably by indiarubber

cups, which render the bottle straight when cooked. Failing this, it can be effectively performed by placing the bottles upright in a jar, the mouth of which is closed by a cork perforated to allow of the admission of a thermometer. The bottles are then covered up to their necks in water, heated up to the required temperature, which is maintained for twenty minutes. In the same way the milk sufficient for twelve hours may be put in a jar closed by a cork, through which is passed a thermometer, and heated in a saucepan of water to 150°F . for twenty minutes.

In whichever way pasteurization is carried out, the milk should be cooled quickly in order to prevent the incubation of spores as far as possible, which is best effected by placing it in running cold water. It should then be kept in a cool, clean place.

Sterilization.—This can only safely be done at home in some such apparatus as is shown in Fig. 7. Each bottle contains one feed, and when filled is covered by a special rubber cap. The bottles are placed in the rack within the sterilizer, and are surrounded with water up to the level of their shoulders. The water is boiled for twenty to forty minutes. On cooling, the bottles become hermetically sealed by their rubber caps. When required a bottle is taken and warmed up to blood-heat in the "food-warmer" provided. Its cap is then removed and a teat slipped on. It is then given to the baby. The cost of most patterns of sterilizers is about fifteen shillings.

By rearing many infants from birth on sterilized milk, Prof.

Budin (who used apparatus similar to that shown here) proved that prolonged boiling renders the milk easily digestible. He also showed that where separate bottles were used, as in this method, there is no risk of producing scurvy, even where the milk is boiled for forty minutes, if milk of good quality be used (p. 24).

By sterilization the taste of milk is altered, but this practically never causes any difficulty in the case of infants under six months of age.

Scalding.—For the poor, who have neither the money to buy apparatus, nor the time to spend upon preparing milk carefully, scalding is a satisfactory solution of the difficulty. By this is meant that



FIG. 7.—APPARATUS FOR STERILIZING OR PASTEURIZING MILK IN SEPARATE BOTTLES. The one shown here holds seven four-ounce bottles, supplied with rubber caps, which seal the bottles on cooling. With it are provided bottles, cups, teats, thermometer, brush, and food-warmer. (Haskelly, Oxford St., London, W.)

the milk is heated until bubbles begin to rise to the surface, which occurs at about 220° F. Scalding may be done by placing the milk in a saucepan and keeping it at this degree of heat for five minutes. A better method consists in placing the vessel containing the milk (if in separate bottles, so much the better) in a saucepan of water, and boiling the water for five minutes. Here, again, the process of cooling should be conducted rapidly.

Frequency of Feeding.—The number of feeds in the twenty-four hours, and the intervals to be observed between them, are the same in the case of hand-fed children as in those at the breast. They have already been given (p. 24), and are included in Table 16 showing a scheme of bottle-feeding (p. 30).

Quantity per Feed.—The best guide to the quantity to be given at each feed is the infant's appetite, assuming that the bottle is correctly given, and the child neither encouraged to take more than it wants nor to desert before it is properly satisfied.

At the same time it must not be forgotten, that the bulk of a feed, as opposed to its composition, is not an unimportant factor in the production of gastric symptoms. It is not always sufficiently recognized that vomiting may be due to feeds that are too large in volume, and not always to the milk-mixture being too strong. In the same way, distention of the stomach may be brought about by giving too bulky meals.

For these reasons, therefore, it is well to remember the capacity of a baby's stomach, and a very simple series of figures is of sufficient accuracy for our purpose. At birth the stomach will hold one ounce, at two months two ounces, and so on, gaining one ounce for each month of life up to the sixth month.

Composition of Cow's Milk.—In the annexed table is shown a comparison of cow's milk—unified and in various dilutions—with human milk. It will be seen that the curd-forming protein, classed as *casein*, in cow's milk, is not reduced to its proportion in human

		HUMAN MILK PER 100 GR.	COW'S MILK PER 100 GR.					
			Two Diluted	1 to 1	2 to 1	3 to 1	4 to 1	5 to 1
Proteins	Casein	0.6	1.25	1.00	0.68	0.81	0.65	2.10
	Lactalbumin	4.4	0.75	0.51	0.25	0.18	0.15	0.5
Fat		3.8	5.5	6.75	1.66	0.81	0.7	2.34
Lactose		5.6	4.0	2.0	1.1	0.0	0.8	2.0
Salts		0.2	0.2	0.33	0.11	0.17	0.14	0.16

TABLE 1.—COMPARISON OF HUMAN MILK WITH COW'S MILK, UNIFIED AND IN VARIOUS DILUTIONS WITH WATER.

milk until a dilution of 1 in 5 (one part of milk added to four parts of water) is reached.

Modification of Cow's Milk for Normal Infants.—The chief difficulty in feeding infants upon cow's milk arises from the excess of casein in cow's milk as compared with human milk: the former containing 3.25 per cent and the latter 0.6 per cent.

We may endeavour to overcome this difficulty (a) by diluting the milk, (b) by rendering the coagulum more easily digestible, as by the addition of sodium citrate, or (c) by a combination of these two methods.

Feeding by Diluted Milk.—This is the method most commonly employed. Before describing it, it may be well to consider for a moment its advantages and drawbacks.

By diluting cow's milk, the excess of casein is certainly diminished to some extent, and thereby the digestion of the milk is more easily accomplished; but the casein of cow's milk is not reduced to its proportion in human milk until a dilution of 1 in 5 is reached. Such a weak milk-mixture as this is not, of course, in use except during perhaps the first few weeks of life, and an infant, therefore, has to become accustomed to taking a milk in which the proportion of casein is higher than it is in human milk. In other words dilution by itself, as generally used, only partially deals with the excess of casein in cow's milk. On the other hand, the proportion of fat is lowered considerably by dilution, and has usually to be corrected by the addition of cream, since in cow's milk the percentage of fat is usually the same as in human milk. The sugar in cow's milk, even when anhydrous, is less than in human milk, and when water is added to the milk this deficiency is so great as to require correction. The salts, as will be seen by reference to the table on p. 33, are excessive in cow's milk, and a dilution of 1 in 4 may be reached before there is any marked deficiency in this constituent.

These additions of water, cream, and sugar, form a drawback to the use of diluted milk. In the first place the bulk of the feed is much increased; not always a matter to be disregarded. There must needs be extra handling of the milk which, in poor surroundings, is not to be encouraged. For the poor, the purchase of cream is a considerable and often impossible expense, although this may be overcome by the use of butter, or of cod-liver oil obtained from a hospital. Cream, especially that sold to poor people, is even less satisfactory in its freshness and purity than milk. Lactose is expensive, and in poor patients its place is usually taken by ordinary brown sugar, which may upset an infant's digestion. Finally, although it is usually easy to give fat in diluted milk in sufficient proportion to prevent the occurrence of rickets (p. 80), yet there is a manifest difference between this and giving a full complement of fat.

The drawbacks to the use of diluted milk have been set down in full, because it is necessary that they should be appreciated in

order that the causes of possible failures may be the more easily traced.

Dilutions.—The dilutions which are most commonly used are those given here, but it must be remembered that they are subject to a good deal of variation, erring rather on the side of being too weak than too strong. At nine months, for instance, many infants take pure milk quite satisfactorily.

During the first few days of life whey, given alone, is a satisfactory food, and a milk mixture (1 in 4) may be started on the third or fourth day. There is, however, no objection to giving this from the first.

The dilutions are given in the following table:

Age	Milk	Diluent
1st week	1	4
2nd, 3rd and 4th weeks	1	3
At one month	1	2
At two months	1	1
At three months	1	1
At six months	1	1
At nine months	1	1

TABLE 1.—DILUTIONS OF MILK, APPROXIMATE TO BE USED AT VARIOUS AGES

The changes are in all cases to be made very gradually.

Dilutents.—The various dilutents and their chief advantages may now be considered.

Whey.—This is theoretically the best diluent for milk. It contains all the soluble protein (lactalbumin), the sugar, and about 1 per cent of the fat of milk. Where it is used, the sugar in the milk-mixture tends to coagulation. In preparing the whey the rennin must be destroyed, otherwise the milk to which it is added will be coagulated. This may be done at a temperature of 150° F., at which temperature the lactalbumin is not coagulated. No harm, however, is done if a higher temperature is used and the lactalbumin changed. The coagulation of this occurs at 160° F.

Boiled Water.—For ordinary purposes, especially in dealing with the infants of the poor, boiled water is the most satisfactory diluent.

Barley-water.—This is the diluent in most general use. It contains as usually prepared from .5 to 1 per cent of starch. As the starch-digesting power of young infants is very small, this may be may not be an advantage. It must be remembered, therefore, that barley-water may set up flatulence and colic, causing malassimilation of the food, and consequent looseness of the bowels. Where a mild laxative effect is advantageous, barley-water is of particular value, but there is no reason for its routine use. Its action in rendering the food more easily digestible is extremely slight.

Rice-water.—This is not much used. It is less laxative than barley-water. It may be prepared by soaking two tablespoonfuls of washed rice in a quart of warm water for three hours: allowing the mixture to simmer for 24 hours, and then straining.

Lime-water.—The chief value of this diluent is that it has some effect in counteracting any tendency to looseness of the bowels: but it also renders the coagulum of casein more flocculent, and to some extent hinders the action of rennet in the stomach. Its power as an antacid is, however, small. It may be used in the proportion of one or two tablespoonfuls (or a quarter to half a drachm of the stronger liquor calcis saccharatus) to every three ounces of milk.

Bicarbonate of Soda.—This is a stronger antacid than is lime-water. It is of particular value where there are colic, vomiting and constipation, such symptoms as may be due in part to acid dyspepsia. It may be added in quantities of two or three grains for each ounce of the feed. Washing and baking soda are less satisfactory than the chemically prepared sodium bicarbonate.

Sodium Citrate.—The use of this important measure is described separately on page 30.

The Addition of Fat.—Human milk and cow's milk both contain a similar average of fat, 3.5 per cent. As soon as water is added to cow's milk there is therefore a deficiency of fat as compared with breast-milk.

While it is undoubtedly the curd-forming constituent of milk that causes the chief difficulty in digestion, and for which dilution is generally practised, the proportion of fat in an infant's food cannot be neglected.

A deficiency of fat in the diet is, as is well known, the most potent and constant factor in the production of rickets. There is, however, little danger of this disease arising if the proportion of fat is not less at the various ages than is the case in the dilutions of milk that are ordinarily employed at the various ages, namely:—

At 3 months 1 of milk to 1 of water	=	5.75 per cent of fat.
— 6 — — — — 1 — — — — 1	=	7.52 — — —
— 9 — — — — 1 — — — — 1	=	2.92 — — —

There is, therefore, plenty of latitude in the matter of fat supply before rickets is induced, although a greater percentage of fat than these dilutions give is beneficial to the child in most cases.

An excess of fat in the food quickly brings about looseness of the bowels, the stools becoming pale and greasy. In this way information is usually quickly obtained that the fat supply is excessive.

As a rule, more than the natural amount of fat in breast milk (3.5 per cent) is not well borne, and it is preferable to give rather too little than too much of this ingredient. We may aim, therefore, at giving between 3.0 and 4.5 per cent, although in a few instances not even as much as 3 per cent can be tolerated.

Cream.—A standardised cream is practically not on the market. The cream sold in London is almost invariably that which has been

prepared by means of a separator or centrifuge, and is known as "separated" or "centrifugal" cream. This contains about 45 to 50 per cent of fat. "Gravity" cream, obtained by skimming milk which has been allowed to stand for twelve hours, contains from 30 to 35 per cent of fat.

For feeding purposes these strengths must be remembered, and according to Dr. Still, the most accurate figures are 45 per cent for "separated" cream (the ordinary cream bought in London), and 32 per cent for "gravity" cream.

Making use, therefore, of separated cream, it is easy to calculate the amount that is required to raise the percentage of fat in the diluted milk to the necessary figure. The quantity of fat added as cream is obtained by dividing the dilution of cream in water into 45; thus, 1 dr. of cream in 3 oz. of water (i.e., a dilution of 1 in 24) gives a 2 per cent proportion of fat. This is the most convenient number to remember, although the others may be easily worked out.

These figures may be tabulated as follows, reckoning the cream as containing 45 per cent of fat:—

	Water added to 10.	Percentage increase of Fat
1 drachm of Cream (45% fat)	0.62	1
	3.02	1.2
	4.62	1.5
	5.02	2
	2.02	5
	1.02	10

TABLE 4.—PERCENTAGE OF FAT IN CREAM AND WATER MIXTURES

By this means a known deficiency of fat in a diluted milk (Table 1, p. 33) can easily be remedied.

In making additions of cream care should be taken to add accurate amounts. A domestic teaspoon frequently holds 2 dr. or more.

Butter.—This means of introducing more fat into a milk mixture is not so satisfactory as the foregoing. Butter, however, is cheaper than cream. Its taste is not usually pleasant to infants, nor does the butter mix well with milk, so that it is more liable than cream to upset the digestion. While both milk and cream are subject to adulteration by preservatives, butter is still more open to this objection. For these reasons, therefore, butter is less suitable for infant-feeding than cream, and consequently a high percentage of fat is best avoided if butter has to be used. Butter may be reckoned as containing 80 per cent of fat. To three ounces of diluted milk (1 to 1) a piece of butter the size of a pea may be added.

Col-lins Oil.—For the very poor a cod-liver oil emulsion may be used, as this can be dispensed from a hospital. Small doses, according

to the infant's age, of a good emulsion are well taken and form a very convenient way of administering fat.

Top-milk Feeding.—This is a method which is sometimes used in order to obtain a high percentage of fat relative to the proportion of proteins. Milk is allowed to stand until the cream has settled at the top, and the upper portion only is used for feeding. If the upper third is used the amount of fat is three times that of ordinary milk; while if the upper half is taken, the amount of fat is twice that of ordinary milk—but in neither case is the percentage of the proteins appreciably altered. It is obvious, therefore, that suitable dilutions of top-milk can be made which are rich in fat as compared with their proportion of protein.

The apparatus used in humanizing milk (illustrated in Appendix A) may be conveniently employed for preparing top-milk feeds.

This method has no advantage over that of adding "separated" cream to a dilution of milk, while it has the disadvantages of requiring the milk to stand for some hours after it reaches the house, to need careful working to necessitate more handling, and of being not very constant in its resultant percentages of fat.

The Addition of Sugar.—Cow's milk undiluted contains only 4 per cent of sugar, while human milk contains 7 per cent. There is then a considerable deficiency of this constituent in any form of diluted milk.

The amount of sugar in the diet is not of the greatest importance as a general rule. Nevertheless, we should aim at giving it in the same proportion as it occurs in breast milk. As an excess of sugar may give rise to flatulence, colic, diarrhoea, and occasionally acute eczema, we shall do well to give less rather than more than 7 per cent in a milk mixture.

In order to do this it is convenient to remember that a 5 per cent solution may be made by adding to three ounces of fluid a level teaspoonful of sugar (using a teaspoon of the usual domestic size, which holds two drachms of water) or a lump of cane sugar half an inch square.

The amount of sugar in the diluted milk being known (by dividing the 4 per cent of normal milk by the number of dilution) the amount to be added is easily calculated. Thus milk diluted 2 in 4 contains 2 per cent of sugar; with the addition of a level teaspoonful of sugar for each three ounces of the fluid, the percentage in the mixture is brought up to 5 per cent.

For most purposes, whatever be the dilution of milk used, the addition of 5 per cent of sugar (a level teaspoonful or a lump half an inch square to every three ounces) gives roughly a satisfactory amount.

It is best to keep the sugar in a dry form and dissolve it when required, as a solution of sugar, if kept, is apt to become contaminated.

Lactose is preferable to cane-sugar, except where expense is a consideration. It is less apt to give rise to flatulence and diarrhoea.

Summary.—The details which have already been given concerning the frequency and the volume of the feeds, together with the dilutions suitable for the various ages, may be summarised in the form of a tabulated schedule. The figures given are to be regarded as approximate only.

AGE	FEEDING, IN TABLETS PER DAY	NO. OF FEEDS IN 24 HOURS (5 MEALS)	NO. OF FEEDS IN 24 HOURS (4 MEALS)	DILUTION		AMOUNT OF MILK IN EACH FEED
				WATER	LACTOSE	
1st to 7th days	2-hourly	2	20	1	1	1-2 oz.
2nd to 4th weeks	2-hourly	2	10	$\frac{1}{2}$	$\frac{1}{1}$	1-1 oz.
3rd month	2-hourly	1	5	1	1	1-4 oz.
4th & 5th months	2-hourly	1	7	1	1	4-5 oz.
6th to 9th months	2-hourly	0	5	$\frac{1}{2}$	$\frac{1}{1}$	5-8 oz.
10th to 12th months	2-4-hourly	0	5	$\frac{1}{2}$	$\frac{1}{1}$	8-9 oz.

TABLE 14.—SCHEDULE FOR INFANT-REARING, FROM BIRTH TO AGE 12 MONTHS.

Feeding by Citrated Milk.—The addition of sodium citrate in the proportion of two grains to every ounce of milk produces a great change in the coagulum formed on digestion, as may be tested *in vivo* or by means of a test meal in an infant. This valuable method of modifying the milk-clot was introduced by Sir A. E. Wright, and first applied to infant-feeding by Dr. Paynton.

By sodium citrate we are then enabled to modify the curd so that it is more easily digested, or in the case of abnormal infants, with which we are here concerned, we are enabled to give a much stronger preparation of milk, modified by the addition of sodium citrate, than would be possible without its use. We see, therefore, that it is in the first place an important aid to feeding by diluted milk mixtures.

It is, however, more than this. Professor Bain, using milk which had been subjected to prolonged boiling, showed conclusively that it was perfectly possible to feed infants upon undiluted milk ("whole milk"). Dr. Langmead has initiated the method of feeding by means of whole-milk modified by the addition of sodium citrate;

and being convinced from an extensive use of this method of its great advantages, I propose here to consider it.

Advantages.—Citratized whole milk has a great advantage over diluted milk, in that the bulk of the food is much lessened. This in a great many cases of both normal and wasted children, is in itself a matter of importance, for time after time it is seen that a small feed is obtained

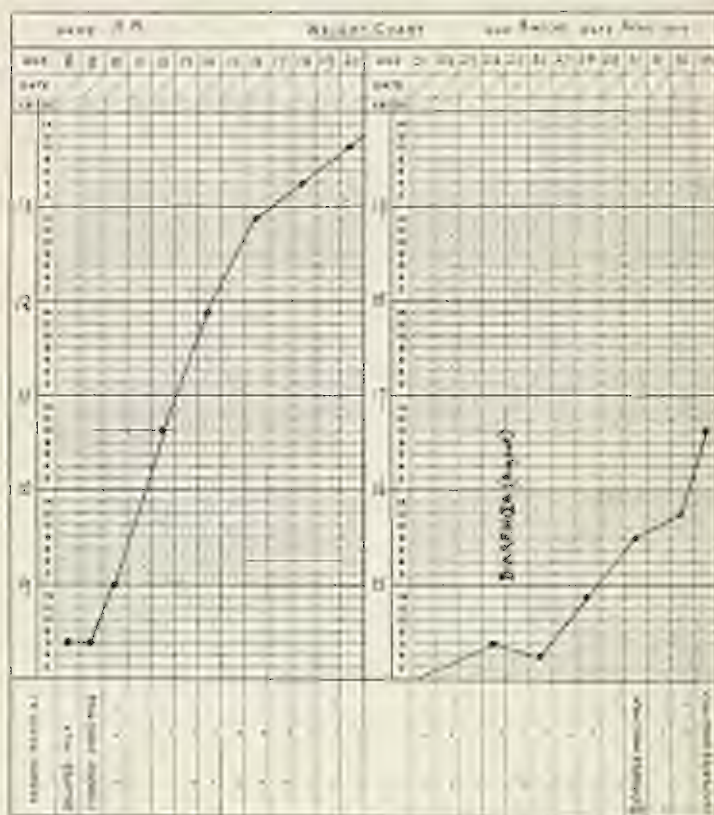


FIG. 8.—WEIGHT CHART, IMPROVED PROGRAMME IN CHILDREN WHO FIRST FEED ON CITRATIZED CREAM MILK.

where a large one is wanted. For the same volume of food, therefore, a much larger amount of nutritive material is given where whole-milk is used than where feeding is by diluted milk. For this reason (a matter of minor importance) there is much less wetting of napkins and consequent crying, than in infants on a milk mixture. Again,

the use of citrated whole-milk is an exceedingly simple and cleanly procedure; while it is considerably cheaper than a milk mixture to which cream is added. These points make it a particularly valuable method of feeding the infants of the hospital classes, where the addition of cream is a great expense and involves extra handling and a great increase in the possibility of contamination. As has been said before, the cream supply of London is in even a sadder state than that of the milk. The muscular development of children reared by this method is extremely good.

Disadvantages.—One disadvantage urged against the use of sodium citrate, with either diluted or whole-milk, is that it is constipating. I am not convinced that this is true, but at all events its action in this way cannot be more than slight, and is easily guarded against. In that constipation is very frequent and arises in breast-fed infants in particular, this cannot be advanced as a very real drawback to its use.

I am quite convinced that the use of whole-milk, when it agrees well with the patient, has no tendency to produce dilatation of the stomach or any of the other ills that have been attributed to it. It may, I believe, after prolonged administration, produce a transient edema, although I have never seen such a result.

Method of Feeding.—The method I use is almost exactly similar to that recommended by Dr. Langmead and, as will be seen, is very simple and clean. A solution of sodium citrate is ordered of such a strength that it contains in a drachm sufficient of the drug for one bottle, in the proportion of two grains to every ounce of milk. This for a three-ounce feed is prescribed as written, *R. Sod. Citrat. gr. vi. Aq. Destill. ad 3j.* At the time of preparation of the feed, a teaspoonful (i. drachm) is put into the amount of milk that is to be used for the feed, and this is then scalded or pasteurized, and when cooled is given to the infant. No addition of fat or sugar is of course required.

Healthy children may be fed by this method after the first fortnight; but as a rule I have not used citrated whole-milk under the age of six or eight weeks. Before this time, a dilution of one part of citrated milk to one or two of water may be used.

By the sixth month it is generally possible to reduce the amount of sodium citrate added, so that the infant takes half citrated milk, and this is gradually further reduced until a pure unmodified milk is taken.

The volume of the feeds will not of course need to be so large as in the case of feeding with diluted milk, and an amount well within the capacity of the stomach at the various ages (p. 33) may be given.

FEEDING AFTER THE NINTH MONTH.

From Ninth to Twelfth Months.—The methods of bottle-feeding and of weaning at the ninth month have been described.

Starch should be added to the diet at about this time. The exact age at which the addition of starchy foods is beneficial is very varying. In some children it is of good effect as early as the sixth month, and occasionally even at the fifth month; in others it is better withheld until the twelfth month. As a general rule it may be said that when one or two teeth have been cut starchy foods are likely to be of value.

Starch may be added in several ways. Boiled bread, in the form of bread and milk, entire wheat flour, plain milk pudding, or porridge made with milk, may be given twice a day. Lists of proprietary foods containing starch partly converted or wholly unconverted are given on pages 51 and 55. When any of these is used it is well to disregard any instructions that may be issued by the proprietors, and administer a couple of drachms in the milk twice a day in the day.

During these months spoon-feeding should be started.

From Twelfth to Eighteenth Months.—At a year old a little more solid food may be given, but two pints of milk should be taken daily. The bottle may now be entirely discarded. Potato and gravy, red meat gravy, broth, bread and butter, bread-crumbs fried in bacon fat, and occasionally the yolk of an egg, may be added to the food already mentioned.

Fruit-juices, which may be given at any time of infancy, may be used with freedom now.

During these months the child receives five meals a day. Some milk may be given during the night if it appears to be needed.

From Eighteenth to Twenty-fourth Months.—By this time the child eats freely with a spoon, and drinks from a cup. Pounded or minced meat (chicken, fish, bacon, or mutton) may now be given. After a little time these are best given finely chopped, so that some mastication is required.

At the end of this period a little green vegetable, such as spinach passed through a sieve, may be tried. For puddings, custard, rice pudding, corn-flour, and purée may be given.

During Third and Fourth Years.—The diet is now to be increased in quantity. Meat, light farinaceous puddings, eggs, and fruit may be given as before, but more regularly and in larger amounts. Green vegetables of various sorts may now be taken. Spinach, cauliflower, cabbage, stewed celery, green peas, passed through a sieve or finely minced, may be given. Raw apples or currants are not to be allowed, but softer fruits, such as bananas, pears, peaches, and the juices of various fruits may be given. Tea and coffee should not be allowed under the age of five years.

After Five Years.—By this time the diet of the child presents no difficulty. Obviously indigestible food must be avoided. Greater attention is now usually necessary towards seeing that the child eats its food properly, masticating it well, that its meals are taken regularly, and that nothing between meals is allowed.

III.—INFANT FEEDING IN DIFFICULT CASES.

For the most part bad results in infant-feeding are the outcome of a lack of observance of the rules already detailed, which should govern the frequency and regularity, the quantity and composition, of the feeds to be given to a baby. Where these are strictly attended to, the infant's progress is usually satisfactory.

In a small proportion of cases, however, the most careful feeding by the ordinary methods results only in indigestion and a failure to gain weight. Most frequently, some past fault in feeding is responsible for this lack of progress: the infant's digestion having been once upset is unable to cope with even an ordinary diet carefully given. In other cases, although these are exceptional, the digestive disturbances appear to be due to an inherent weakness of the powers of digestion and assimilation.

Where an ordinary diet is not well borne, it is evident we must have recourse to some modification of the food, by which it is rendered more suitable for the case under treatment. Our first step in this direction must be towards ascertaining in what way the diet is unsuitable, why it is that the method of feeding which answers well in most cases fails in some particular instance.

Failure to gain weight on an ordinary diet is usually due, as far as the feeding is concerned, to one or more of the following reasons: (1) *The bulk of each feed may be too large*; (2) *The infant may not be able to digest the fat in the food*; (3) *It may not be able to digest the curd of the milk*. Of these, the third is the most important, for curd-indigestion is the commonest of the difficulties met with in the artificial feeding of infants. The other possibilities must not, however, be overlooked, and for this reason are here mentioned first.

1. Excessive Volume of Feed.—Engelmann has elsewhere based his opinion upon the fact that a not uncommon fault in infant-feeding lies in the administration of a feed of excessive bulk; but we are here concerned with those instances in which a quantity suitable for ordinary cases appears too large to be tolerated. Such may be suspected to be the case where, almost immediately after a feed, the entire contents of the stomach are vomited. The symptoms closely simulate those of acid dyspepsia, and it is not improbable that in both conditions some spasm of the pylorus is present.

Treatment.—The bulk of the feed must be reduced, and in order that sufficient food shall be given, it is usually necessary to reduce the volume of the feed by lessening the amount of the diluent added to the milk. By thus increasing the strength of the feed there is a risk of setting up curd-indigestion, and for this reason the addition of sodium citrate, in the proportion of two grains to each ounce of milk, is to be recommended.

As a rule, cases of this type progress very satisfactorily on titrated milk, given slightly diluted or whole. The details of this method of feeding have already been explained (p. 35), and I need here only emphasize once more its extreme value in the feeding of delicate or wasted infants who vomit. The progress made by an infant who had wasted on a diet of diluted cow's milk is shown in *Fig. 8* (p. 45).

2. Fat-indigestion.—Where fat is not given in excessive quantities it is usually well digested. Occasionally, however, the amount of fat in an ordinary milk-mixture is too great for an infant's digestion.

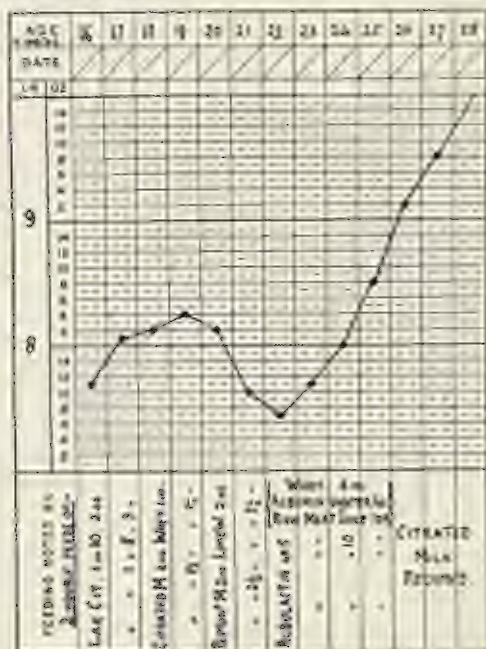


FIG. 8.—WEIGHT-GAIN OF A 3-MONTH INFANT WITH CHRONIC ACUTE DIARRHOEA.

The chart starts when milk is introduced into the diet after severe acute diarrhoea. Day 10 is diarrhoea again set up and periods with loss of weight on modified milk diets. On a diet containing no curd and practically no fat, the diarrhoea ceases and there is a gain of weight. Still feeding is then successfully resumed.

This is particularly likely to be the case where the patient is recovering from an acute attack of diarrhoea. Where this is so the stools become loose, greasy, and pale or green in colour, and contain much fat.

Treatment.—The amount of fat in the diet must be reduced for the time being. Care should be taken to ascertain if the child is being fed upon a milk which is abnormally rich in fat, as in the case in some "merry" milks.

Where no fat can be tolerated a diet containing whey, albumen-water, raw meat juice and albumin may be given (fig. 4).

3. Curd-indigestion.—The curd of cow's milk differs both in quantity and quality from that of human milk, and it is from the excess of an indigestible curd in the former that most of the difficulties in the artificial feeding of infants arise. The proportions of casein in cow's milk and human milk have already been compared, and the methods of dilution ordinarily adopted in order to reduce the amount of curd-forming protein in cow's milk have been detailed. On page 35 is given a table showing the percentage composition of cow's milk in various dilutions with water. It is seen there that it is not until a dilution of 1 in 5 is reached, that the amount of casein in cow's milk is reduced to a figure similar to that of its occurrence in human milk.

It may be pointed out again here, that although cow's milk contains an excess of casein, it contains only half as much of the soluble protein, lactalbumin, as does human milk. Any dilution of the cow's milk will render this deficiency still more marked.

Curd-indigestion may be the result of past feeding on a milk-mixture containing a quantity of casein too great for the infant's powers, by which means its digestion has been so upset that it cannot deal with an amount of curd that is usually well taken by a baby of similar age. On the other hand, there is a certain small proportion of infants whose digestive powers are naturally very weak, in whom the difficulty of digesting the curd of cow's milk is extremely great.

Where curd-indigestion is present, there appear in the stools small masses of curd, and the child may suffer from diarrhoea, vomiting and colic, while the general progress, as estimated by the body-weight, is very unsatisfactory.

We have now to consider the means by which we can overcome this tendency to curd-indigestion in those infants who are peculiarly unsuited to it.

Further Dilution of Cow's Milk.—Evidently an excess of curd-forming protein may be reduced by the addition of an extra amount of diluent. Nevertheless this is a method which has its limitations.

Under ordinary circumstances the infant is able to digest in a diet of diluted cow's milk, a larger proportion of casein than occurs in human milk. Where there is a great difficulty in curd-digestion, and we rely only upon dilution to overcome it, in order that the percentages of casein in cow's milk may be equal to that of human milk, a dilution of 1 in 5 is necessary. By the time this point is reached there is a great deficiency in fat (6.7 per cent), sugar (5.8 per cent), and lactalbumin (0.15 per cent). Thus, while the bulk of the food is increased its nutritive value is much reduced, and fat and sugar have to be added. These are considerable drawbacks to the use of freely diluted milk, particularly in the case of very poor patients, where cream or butter cannot be easily bought.

The value of the method of dealing with curd-indigestion by further dilution may be summarized as follows. In the first place, where further dilution to a slight degree only is required this is a most useful measure, particularly where the circumstances of the patient allow of the use of whey (as the diluent) and of cream. Secondly, where full dilution (1 to 5) is requisite, this method is best confined to cases in the first few weeks of life, at a later age reliance being placed on a slighter dilution combined with some method of modifying the curd, such as citration.

Additions to Freely Diluted Milk.—Where the use of freely diluted milk is more than temporary, fat in the form of cream must be added, as laid down on page 36. Sugar must also be added (p. 36), unless whey is used as the diluent for the milk.

The easily digestible protein, lact-albumin, is present in undiluted cow's milk in less quantity than in human milk (50.75 per cent. and 1.4 per cent. respectively). Dilution with water makes this deficiency of this valuable food-stuff become still more marked. This loss may be avoided by using whey as the diluent, as this contains all the lact-albumin of cow's milk. A preparation of lact-albumin under the name of "Albumin" has quite recently been put on the market (Wiffin & Co.). Further trial of this preparation is as yet necessary, but there is no doubt that, as a soluble protein, it is of considerable value in aiding the feeding of infants upon freely diluted milk.

Citrated Milk.—One of the most successful means of dealing with curd-indigestion is by the administration of citrated milk. The addition of sodium citrate in the proportion of two grains to each ounce of milk so profoundly modifies the curd that its digestibility is much increased.

In very delicate children, citration may be combined with free dilution; but as a rule, citration enables a strong milk mixture, or even undiluted milk, to be well taken.

The methods of preparing and using citrated milk are given on page 42.

Peptonized Milk.—By peptonization, the curd of cow's milk is rendered more easily digestible. The methods of preparation are given in Appendix A.

The value of peptonization in increasing the digestibility of the curd depends upon the length of time during which the enzyme is allowed to act in the preparation of the milk. Where the milk is peptonized for the ordinary period of ten to fifteen minutes, only a comparatively small proportion of the protein, usually less than a third, is altered, and this offers no great advantage over milk fully and properly citrated.

With more prolonged peptonization a greater proportion of the protein is altered, but unfortunately a better taste is given to the milk, so that an infant may refuse it altogether. Many, however, take it well.

is sweetened, in which case such a preparation may be of great value as a temporary measure in overcoming curd indigestion.

Scoury has occasionally arisen on a diet of peptonized milk, so that care should be taken on this score. It is sometimes said that the use of predigested milk tends towards atrophy of the gastric glands, but there seems no foundation for such a fear.

In passing from peptonized to unpeptonized milk the change must be made very gradually. The length of time to which the milk is subjected to peptonization is slowly reduced; then mixtures of peptonized and ordinary milk are given until the predigested milk is withdrawn altogether. The change usually presents no great difficulty if made sufficiently slowly.

Milk Subjected to Prolonged Boiling.—The boiling of milk for thirty or forty-five minutes produces changes in the curd which render it more easily digestible. Although there is probably no danger of producing scoury if the milk be boiled in separate bottles, each containing enough for one feed (p. 32), this method offers no advantages over citration as regards the increased digestibility of the curd, while it is clearly less convenient.

"Humanized" Milk.—Many dairies sell sterile milk preparations of constant composition under the name of "Humanized milk." Some of these are prepared by a complicated process which cannot well be undertaken at home, but are of great service as the treatment of mild cases of curd indigestion. Before ordering them, however, it is wise to ascertain the exact composition of the preparation it is proposed to use.

The method of preparing humanized milk at home is given in Appendix A.

Welford & Sons prepare two varieties of "humanized milk," each at the price of 7d. per pint, delivered in half- or one-pint bottles; 5-oz. bottles are supplied at a slight extra cost if ordered. The percentage compositions of these, as given by the makers, are as follows:—

	"Flemish" Humanized Milk.	"Milkmaid" Humanized Milk.	Express Milk (the composition.)
Protein	5.33 per cent.	5.91 per cent.	2.0 per cent.
Fat	3.60 "	5.60 "	1.5 "
Lactose	6.60 "	6.40 "	7.0 "
Salts	0.54 "	0.23 "	0.8 "

Table 11.—WELFORD & SONS' HUMANIZED MILK PREPARATIONS.

The Express Dairy Company supply somewhat similar preparations, delivering them in bottles each containing sufficient for one feed only, and suitable for use as feeding-bottles with the application of the necessary test. The cost of feeding an infant on these products is

This may be given as suppers per day. The following "approximate" analysis of their preparations is given by the makers:—

	PREP. A.	PREP. B.	PREP. C.
Protein	1.0 per cent	1.3 per cent	1.5 per cent
Fat	1.0	3.73	4.0
Lactose	1.0	0.75	1.5

Table 11.—THE LARSEN DAIRY CO.'S BROOKLINE, MASS. PREPARATIONS.

Desiccated Milk.—Preparations of milk dried without the addition of malted cereals have been recently introduced. One of the best of these is sold under the name of "Glaxo": in its manufacture cream and milk sugar are added to milk, and the mixture dried and packed in the form of a fine powder. When diluted for use, the fluid contains a relatively low proportion of protein and a high percentage of fat. The coagulum formed during digestion is of a fine granular type, so that some infants who cannot digest ordinary cow's-milk are able to take this preparation well. For feeding the children of the very poor a desiccated milk such as "Glaxo" has the advantage that by this means a sterile milk of good quality can be supplied them from a dispensary or hospital.

Instructions for the preparation of the "Glaxo" feeds are boxed with the milk powder. It is best for infant feeding that each feed should be prepared separately. The milk powder may be used in dilutions in water of 1 in 8 or 1 in 10. In the latter dilution it costs about 3d per pint. The following analyses, made by Mr. H. D. Richmond, have been supplied me by the "Glaxo" Company.

	"GLAXO"	1 in 10	1 in 8
Protein	19.0	1.90	2.31
Fat	27.4	2.74	3.37
Milk sugar	38.0	3.80	4.75
Mineral matter	1.5	0.45	0.94

Table 12.—COMPOSITION OF "GLAXO" FEEDS (DILUTED 1 in 10).

Another desiccated milk is sold under the name of "Eak Cit." It is a sterilized humanized milk, desiccated, and is stated by the makers to have the following composition: Protein 2.7 per cent, fat 2.5 per cent, lactose 16.4 per cent, and ash 5.6 per cent. It may be used in the same dilutions as "Glaxo." It forms a particularly finely divided coagulum on artificial digestion. Its cost is much the same as that of "Glaxo."

I HAVE NEVER SEEN OR HEARD OF SCARVY arising from the use of these

preparations, although it is of course said to be a possible result. Such a danger is easily avoided.

Ass's Milk.—As a temporary measure ass's milk may be of very considerable value in a few cases of severe curd-indigestion. As is seen in the accompanying table, it is a very weak milk, while its curd is firmer than that of cow's milk, even when diluted to a corresponding degree.

		COW'S MILK	ASS'S MILK	HUMAN MILK
Protein	Casein	3.75	1.0	0.0
	Lactalbumin	0.75	0.8	1.4
Fat		3.2	1.0	3.5
Sugar		4.0	3.5	7.0

Table 11.—THE COMPOSITIONS OF COW'S, ASS'S, AND HUMAN MILK COMPARED.

In use, ass's milk should not be boiled, but merely warmed up to the temperature at which it is to be given. It should not be diluted.

It has several disadvantages. It produces a slight laxative effect; it is so weak a food that its use cannot be continued with advantage beyond two or three weeks, even in the case of very delicate children. Lastly, its cost in London is about three shillings per pint.

Wet-Nursing.—The subject of wet-nursing has already been dealt with (p. 29), and is only mentioned here in order to emphasize the fact that, in a few cases, breast-feeding by means of a wet-nurse gives the infant its best or even its only chance of survival.

Whey and Cream Mixtures.—Up to the present we have been considering the methods of feeding infants on fluids containing some proportion of curd-forming protein; but in the method we have now to study the child is fed upon a fluid containing no curd.

Whey contains the lactalbumin, salts and sugar of cow's milk, together with an amount of fat varying from a trace up to about 2 per cent. The amount of fat in whey depends upon the richness of the milk from which it is prepared and the method of preparation (see Appendix A).

The percentage compositions of cow's milk, whey, and human milk are compared in the accompanying table:—

		COW'S MILK	WHEY	HUMAN MILK
Protein	Casein	3.75	Nil	0.0
	Lactalbumin	0.75	0.75	1.4
Fat		3.2	Up to 1.0	3.5
Sugar		4.0	4.0	7.0

Table 12.—COMPOSITIONS OF COW'S MILK, WHEY, AND HUMAN MILK COMPARED.

Whey, although free of curd-forming protein, is seen to be deficient in fat-solvent, fat, and sugar, as compared with human milk. These deficiencies can, however, easily be rectified. The addition of cream and of lactose in the proportions of one drachm and half a drachm respectively to each three ounces of whey, corrects the lack of fat and sugar. The deficiency of protein may be corrected by the addition of raw meat juice (a drachm to each 3 ounces), albumin or other protein preparations.

By these means, therefore, we can produce a food which resembles human milk even in the absence of curd-forming protein. Further, its composition is easily altered at will to allow for any special needs of the case.

In theory, then, for the feeding of delicate children a whey and cream mixture has everything to recommend it; but in practice it is found that, although often of the greatest value, it scarcely fulfils all that might be expected of it on theoretical considerations. Children seldom thrive on it for very long, and as a general rule to which there are exceptions, it may be stated that the use of a whey mixture for longer than two or three weeks is not likely to be beneficial. As a temporary measure this method of feeding may be of the utmost value, while a whey and cream mixture makes an excellent medium through which the administration of milk may be gradually resumed.

Condensed Milk.—The preparations which are sold as condensed milk are made of milk which has been concentrated to roughly one-third of its original bulk. Thus, if a full-cream milk has been used and no additives have been made to it, by adding water in the proportion of one part of condensed milk to two parts of water (a dilution of 1 in 3), we should obtain a fluid of similar composition to cow's milk. In order to suit this to the needs of an infant's digestion, it will require further dilution, together with the addition of cream and sugar, just as is necessary in the case of fresh cow's milk.

These then are the principles underlying the preparation and use of condensed milk; but in the case of many of the brands on the market other points have to be considered. The milk, for instance, may have been modified in various ways previous to condensation, and other substances, usually sugar, may have been added to it.

Condensed milks may be divided into three classes:—(1) *Unsweetened brands made from full-cream milk*; (2) *Sweetened brands made from full-cream milk*; and (3) *Cheap brands made from skimmed milk*.

The last class, made from milk from which part of the fat has been removed before condensation, may be quickly dismissed. It should not be selected for use in infant feeding.

1. *Unsweetened Condensed Milk*.—Theoretically, this is the best type of condensed milk, but for various reasons to be mentioned later, the unsweetened brands are not so popular as the sweetened, and consequently are less easily obtained.

The "Ideal" brand (Nestlé) may be quoted as a good example of

this type. It contains protein 8.3 per cent, fat 12.4 per cent, and lactose 16.0 per cent. Evidently we can prepare dilutions of various strengths which will contain a good proportion of fat and no excess of protein. The addition of extra lactose will be necessary. Thus, a 1 in 4 dilution will contain protein 2.08 per cent, fat 3.1 per cent, lactose 4.0 per cent. To this, lactose may be added to bring the percentage of sugar to 5 or 7 per cent (p. 38).

Nestlé's "Viking" brand, also unsweetened, contains protein 9.0 per cent, fat 10.0 per cent, and lactose 13.1 per cent.

A more suitable preparation—more suitable because condensed milk is only to be used in cases of curd-indigestion—is that sold under the name of "Humanised," by the Aylesbury Dairy Company. This is a condensed humanised milk, containing a low proportion of protein. The composition of this, undiluted and diluted, is shown and compared with human milk in the following table:—

	"HUMANISED"		HUMAN MILK
	Undiluted ¹	Diluted 1:1 ¹	
Protein, per cent	8.44	4.22	2.0
Fat ..	10.26	5.13	5.3
Lactose ..	17.31	8.65	7.0

¹ ANALYST'S FIGURES.

² LACTO ANALYSIS.

NOTE 12.—"HUMANISED" COMPARED WITH HUMAN MILK.

Evidently, we have here a very successfully prepared brand of condensed milk: it is, however, rather expensive.

2. *Sweetened Condensed Milk.*—These brands are more popular than the unsweetened preparations, because they are cheaper to use. The added cane-sugar enables the milk, when the tin is opened, to be kept for several days: without it the milk cannot be kept for more than thirty-six hours. Further, a very dilute solution of the condensed milk is used, and there is no need to add sugar at the time of mixing.

The cheaper brands of condensed milk are very deficient in fat, as has already been mentioned, and are worthless for the purpose of infant-feeding.

The best and most popular of these preparations is known as Nestlé's Swiss Milk,* and by this name is meant the "Nest" brand of this company. This contains protein 9.7 per cent, fat 13.7 per cent, sugar (of which two-thirds is cane-sugar) 52.2 per cent. This is, as a rule, used in the proportion of one teaspoonful to six tablespoonfuls of

* The Condensed Milks made by the Nestlé and Anglo-Swiss Co. are Unsweetened, the "Ideal" and the "Viking" brands; Sweetened, the "Nest" brand ("Nestlé's Swiss Milk") and the "Milkmaid" brand. The analyses given are from "Analyses of Foods and Drugs," by Pelletier and Moore.

water. When a teaspoon of the usual domestic size of two fluid drachms capacity is dipped into the milk, it abstracts about three drachms, owing to the quantity which clings to the under surface of the spoon (Stillé). With the above proportions, therefore, we shall be using a dilution of 1 to 8 (1 in 8), and the mixture will contain protein, 1.0 per cent; fat, 1.5 per cent; sugar, 5.8 per cent.

The same company's "Milkmaid" brand contains protein .97 per cent, fat 11.6 per cent, sugar 5.3 per cent.

When we consider the relative proportions of fat and sugar in these sweetened preparations (1 to 4 or 5), it is clear that no simple dilution with water will suffice by bringing them even approximately to the proportions in which they occur in human milk (about 1 to 2). It is evident there must be either a deficiency in fat or an excess of sugar.

The advantages of condensed milk are not numerous. It may be of value inasmuch as it is more easily digested than fresh milk. Condensed milk is usually administered very dilute, and often, as is well-known, it agrees with an infant because of its very weak composition, where cow's milk is said to have failed. But strength for strength, condensed milk is more easily digested than is fresh cow's milk, although in this matter it has little advantage over vitiated or peptonized milk. Another advantage which is claimed for it is its alleged cheapness. This, of course, is a fallacy. Milk which has been subjected to the costly process of condensation, to say nothing of being widely advertised, cannot reasonably be sold at the same price as fresh milk. The apparent cheapness of condensed milk (where it is apparent*) is merely due to its diminished fat-content when mixed for administration, for the value of all forms of milk depends upon the proportion of fat they contain; nevertheless, if the necessary fat can be obtained in the form of cod-liver oil from some charitable institution, the use of sweetened condensed milk relieves poor people of a good deal of expense. Under such circumstances, the supply of a good condensed milk would be preferable. Another advantage urged on behalf of condensed milk is its sterility. It is quite true that the preparation (until when freshly opened is sterile, but this does not necessarily ensure the child obtaining a sterile food. There are many possibilities of contamination before the food actually reaches the baby.

The disadvantages of condensed milks are evident from what has been said on the subject of their composition. Unless a good price is paid, there is a grave risk of inducing rickets by the use of a food which is too poor in fat and too rich in sugar. In the use of any form of condensed milk there is a risk of producing scurvy.

* The mother of an infant, which, although only 3 months old, showed well-marked signs of rickets, informed me that she paid every week 2s. 6d. for condensed milk and 8d. for "grape-water."

The use of condensed milk may therefore be set forth as a very few words. In the first place, it may be of temporary use in the feeding of a delicate child who suffers from acid-indigestion. For this it is not preferable to curdled, peptonized, or desiccated milk, unless used very dilute, when it becomes almost a substitute for whey. Secondly, it may, in circumstances of great poverty, be used where the necessary additional fat is supplied from some charity. As has been mentioned, to supply a good desiccated milk would be preferable. Thirdly, where fresh cow's milk cannot be obtained, as in the case of travellers, condensed milk may be used; but here again a desiccated milk is as good or better.

Patent Infant Foods.—Apart from the preparations of desiccated and condensed milk which have been already considered, there is an enormous number of proprietary articles sold as infant foods. So numerous are these that it is quite impossible to attempt to remember their respective compositions, but it is convenient to bear in mind some rough scheme of classification so that the type to which any one of the better-known foods belongs may be remembered.

In the Tables which are given here, these foods are divided into types, the analyses being only for the purposes of illustration and reference. The figures given are those supplied by the makers of the various brands, or those which have been published by Dr. Robert Hutchison.

These infants' foods are usually divided into two main classes

Class 1.—Foods intended as substitutes for fresh cow's milk.

Class 2.—Foods intended as additions to fresh cow's milk.

CLASS 1.—Foods intended as Substitutes for Fresh Cow's Milk.—These consist of dried cow's milk to which cereal has been added. This class is divisible into two groups, according to (a) the absence, or (b) the presence of unaltered starch.

GROUPS IN CLASS 1.	FOODS	Protein (per cent.)	Fat (per cent.)	Carbo- hydrate (per cent.)
(a) Starch entirely converted	Solt's Soluble Milk Food No. 1	14.5	18.4	52.7
	Allenbury No. 1	10.7	10.7	56.6
	Allenbury No. 2	10.2	14.6	65.2
	Solt's Soluble Milk Food No. 2	16.7	18.3	65.0
	Hestick's Malted Milk	13.0	8.4	51.3
(b) Starch partially converted	Mellin's Infant Food	8.7	3.6	75.9
	Milo Food (Nestlé)	64.0	3.2	75.8
	Carnrick's Soluble Food	11.0	2.5	70.2
Dried Human Milk (for comparison)		12.2	20.4	57.4

Table 12.—DRIED FOODS, CLASS 1, INTENDED AS SUBSTITUTES FOR FRESH COW'S MILK, ANALYSES IN ORDER OF FAT PERCENTAGE.

In the table, the percentage compositions of some of the more important of the foods in Class 1 are shown, and are compared with that of dried human milk, the standard to which they should conform. A glance shows that in all these preparations there is a deficiency of fat and an excess of carbohydrate. Even if the lack of fat be compensated for in use by suitable dilution (where this is possible), the excess of carbohydrate remains. On the other hand, if by free dilution the proportion of carbohydrate be reduced to its proper figure, the deficiency in fat becomes very marked. The ratio of fat to carbohydrate in human milk is approximately 1 to 2, and it is evident that such a ratio cannot be attained by the simple dilution of any of the foods under consideration.

In the foods in Group (a) this excess of carbohydrate is the chief drawback to their use. It is a fault which is no way countered by the fact that they contain no unaltered starch when administered.

The foods in Group (b) contain unaltered starch, and are therefore unsuitable as a general rule for children under the age of six or eight months.

While healthy children have been reared upon some of the foods named, their deficiency in fat and their excess of carbohydrate tend to favour the onset of rickets. All of them favour the production of scurvy, a fact of which the makers are now well aware, as is shown by the directions issued counselling the use of fresh fruit juices. This is probably due to the alteration which the alkaline salts undergo during the preparation of the dried food. Another drawback to their use is their cost, which is relative to the amount of nutriment contained, especially higher than in the case of cow's milk.

Class 2.—Foods intended as Additions to Fresh Cow's Milk.—These are practically cereal foods, and may be divided into three groups:

- (a) Those containing, when mixed for administration, no unaltered starch;
- (b) Those in which the starch is partially converted; and
- (c) Those containing much unaltered starch.

In certain of the foods in this Class, dextrinization of the starch occurs during the mixing of the food for administration. Such are not to be preferred to those in which the dextrinization has taken place during the preparation of the dried product.

Group (a).—The foods in this group are free of unaltered starch, and consequently there is no objection on this ground to their use as additions to cow's milk in the case of infants. Nevertheless, their sphere of legitimate use is small. Occasionally, during the period immediately preceding that of dentition, there is a difficulty in getting the infant to digest a sufficiency of cow's milk, and here the foods may sometimes be supplemented by small quantities of a fully dextrinized food, such as Mellin's or Hovis No. 1, with benefit.

Mellin's food, for instance, is sometimes of value in aiding the digestion of cow's milk, and has a mild laxative effect as well. When used, however, a smaller amount than that suggested in the maker's

directions should be added. One heaped teaspoonful (a domestic teaspoon of two fluid drachms capacity being used) means the addition of 5 per cent of carbohydrate to 3 oz. of a milk mixture (Still). This then constitutes the maximum proportion in which this food should be used.

An excess of carbohydrate in the diet, anything much above 7 per cent, which is the proportion in human milk, is liable to produce colic and looseness of the bowels, and by thus preventing the proper assimilation of fat, may predispose to the onset of rickets.

Groups (b) and (c).—The foods in these groups contain unaltered starch; in the former it is small in amount, while in the latter it is

GROUP OR CLASS II.	Food	Protein per cent	Fat per cent	Carbo- hydrate per cent
(a) Starch entirely converted	Mellin's Food	7.4	Trace	82.0
	Hoar's Baby Food No. 1 ..	7.7	0.3	85.6
	Chalmers Maltose Food ..	5.3	0.2	82.6
	Moseley's Food * .. .	11.8	0.9	75.4
(b) Starch partially converted	Atterbury Malted Food No. 1*	6.2	1.0	82.8
	Benger's Food* .. .	10.4	1.4	79.3
	Savery & Moore's Food*	10.3	1.4	81.7
(c) Starch mostly unaltered	Hoar's Baby Food No. 2 ..	5.2	0.4	90.1
	Robinson's Patent Farley ..	3.1	0.8	83.0
	Hobbs' Escents .. .	14.8	1.0	79.0
	Newer's Food .. .	10.5	1.0	86.2
	Hodge's Food .. .	9.2	1.0	84.7
	Pease Food .. .	15.4	1.2	77.6
	Chapman's Whole Food ..	9.4	1.0	79.5

* Some decomposition occurs during the mixing for administration.

Table 11.—FOODS (Class II.) DIVIDED AT ARBITRARY TO GIVE MILK.

present in considerable quantities. The use of these foods is therefore to be confined as a general rule to infants who are over the age of six or eight months, or who have cut one or two teeth. At this age starch-digestion can be performed, and some of the foods here mentioned may be of use in introducing starch into the dietary, one or two foods being given daily at first. There are of course other methods by which this may be done.

Reference has been made elsewhere [p. 42] to those occasional instances in which, at an earlier age than usual, a small addition of starch to the diet is of benefit. If it is desired to make such an experiment, one of the foods in Group (b) may be tried.

In dealing with patent foods for infants, I have endeavoured to point out to what uses they may legitimately be put. Unfortunately the subject can hardly be left there. Inasmuch as they have their

uses, although they can do little that cannot be done by other means, we as doctors cannot condemn them unconditionally. But when we remember (and who has the opportunity of forgetting?) the vast amount of suffering for which they are responsible, the numberless cases of rickets and other diseases and disorders which they produce, we cannot regard them otherwise than with hostility. We cannot neglect the fact that to order a patent food for one baby may be but to cause it to be used for others. To the lay mind, the food that does one baby good is a "good food," and should therefore suit all other infants; that it was selected with care for temporary use in one particular case for some particular reason, is a fact that is unappreciated or suppressed, and so the alleged excellence of the preparation is raised abroad and the ill-effects of its use are multiplied.

It is therefore adopting no narrow attitude of undue hostility when we regard these foods as articles to be ordered as seldom as possible, and be it remembered, the more fully we understand the methods of modifying cow's milk for infant feeding, the less often shall we be inclined to make use of a patent food.

SUMMARY OF THE METHODS OF FEEDING, AND OF THE TREATMENT OF DIGESTIVE DISORDERS IN DELICATE INFANTS.

We have discussed in detail the rules governing the feeding of normal infants and the various ways by which difficulties in infant feeding may be overcome. I propose now to summarize the matter in a more practical way, passing the various methods in rapid review, comparing and contrasting them, and indicating the general lines upon which difficulties in feeding and disorders of digestion may be dealt with.

Breast-feeding.—The commonest disorders met with in breast-fed children are constipation and colic.

Constipation, which usually co-exists with the colic of these infants, may, if of slight degree, be treated by small doses of olive oil, ipecac, or—a measure of greater utility—repeated doses of glyster powder. If more severe, an infusion of one or two senna pods may be given at night, or a mixture of sodium sulphate, cascara, belladonna, and tinct. vomica may be given once or twice daily. Abdominal massage is of great value (p. 277). Other measures are described on p. 279. At the same time, care must be taken to ensure the foods being given regularly and slowly.

Colic may be treated by the administration of some sodium bicarbonate or citrate just before the infant is put to the breast. Two or three grains of the former, or rather more of the latter, may be given. Carminatives may also be ordered (p. 28).

Vomiting is most commonly due to the child being fed too rapidly, or being given too much at a time. The administration of soda before each feed may be tried. Constipation should be treated.

Lack of progress may be due to the gastro-intestinal conditions mentioned, and attention should first be directed to such a possibility. Should they resist ordinary treatment, or should the child fail to gain weight when the alimentary tract has been put in order, attention must be paid to the condition of the mother's milk. The amount and composition of this may be altered in the ways mentioned on p. 28.

Should we fail by these means to improve the child's weight, in the absence of such gastro-intestinal disturbances as can be corrected, it will be necessary to adopt mixed feeding. The baby should receive as many feeds from the breast as the mother can satisfactorily supply, and these should be supplemented by bottle-feeds containing the amounts and dilutions of cow's milk proper for the age of the child (p. 79).

Unless the mother's milk improves, it will be necessary in all probability gradually to wean the child completely.

Artificial Feeding. The infant should be fed according to the rules which have been laid down for the feeding of normal infants (Table 10, p. 79).

Should the child show symptoms of indigestion, the first thing is to make certain that these rules are being correctly followed. There is no need to repeat here what has been said about the frequency, regularity, quantity, and composition of the feeds, but a few points liable to be overlooked may be mentioned. Be sure that the teat of the bottle is not the cause of the symptoms (p. 79), and that the infant's mouth is properly cleaned after each feed. Ascertain from what feed the milk is being supplied. Have nothing to do with nursery milks or that from one cow; such are prone to vary in their composition, and may be far too rich in fat. Trust, therefore, on having a mixed milk from a reliable dairy. Remember that milk delivered in a sealed bottle *may* have been poured into this from a can in the street, and that a high price does not necessarily mean a good milk—additional reasons for employing a reliable firm.

Assuming that the child is being correctly fed in the way usually suitable for a normal infant, and yet it suffers from gastro-intestinal indigestion, or fails to gain weight, we have to consider the possible causes at work and how to counteract them.

Diarrhoea is the most frequent symptom of indigestion in bottle-fed babies. It may be due to an excess of casein, fat, or carbohydrate, relative to the child's digestive capabilities. With casein-indigestion, the motions contain small masses of white curd, and are loose and often green. Case is often present. With fat-indigestion, the stools are greasy and pale yellow or green in colour. An excess of carbohydrate is not likely to be the cause of the diarrhoea if the child is being fed according to rule. Occasionally, however, even the barley-water used as a diluent to the milk may set up diarrhoea, and whenever the bevels are loose this is best replaced by lime-water or plain boiled water.

In treating this symptom we have, then, first to look to the composition of the food, and to make such alterations as may be advisable. In addition, an aperient such as castor oil may be given, and followed by the regular administration of a castor-oil mixture until the motions begin to improve. The following may be relied on:

R. — <i>℞</i> . Ricin.	<i>℞</i> ss	Tragacanth.	<i>℞</i> ss
Tr. Khol.	<i>℞</i> ss	Aq. Menth. Pip.	ad 5i
Glycerin.	<i>℞</i> ss		

This may be followed later by a bicarbonate and soda mixture. In severe cases, the rectum should be washed out with warm saline, the child being given no milk, but albumen-water or whey. Milk is to be very gradually resumed as the child improves.

Flatulence and Colic in bottle-fed babies are usually associated with diarrhoea, and consequently the treatment given above may be necessary. Warmth to the abdomen should be ordered. A good carminative mixture is such as the following:—

R. — <i>℞</i> . Sod. Bicarb.	<i>℞</i> ss	Glycerin.	<i>℞</i> ss
Sp. Anissa. Aromat.	<i>℞</i> ss	Aq. Cam.	ad 5i
Sp. Chloroform.	<i>℞</i> j		

Colic is recognized by the relief of the pain given by the passage of flatus or feces, and thus is distinguishable from other common causes of screaming, such as hunger, thirst, cold feet, wet nappies, hyperaeridity of the stomach, or the causeless screaming of epilepsy.

Vomiting may be associated with diarrhoea. Assuming that the baby is not fed too quickly, it may also be due to the excessive volume of the feed: in this case, care must be taken that the child is not urged to swallow more than it wants, while the bulk of the feed may well be reduced. In order not to lessen the nutritive value of the food, the amount of the diluent must be reduced, and therefore it is usually wise to citrate the milk (p. 36) to prevent curd-indigestion. Citrated milk is particularly valuable in the case of infants who vomit immediately after taking the bottle.

1. Where the vomiting is severe, the stomach should be washed out (Appendix A). The possibilities of acid dyspepsia and of hypertrophic pyloric stenosis must be borne in mind (p. 372).

Constipation is not a very common disorder in bottle-fed infants. Mild cases may be treated by the addition of cream or cane-sugar to the diet, or by the administration of olive oil in doses of half to one teaspoonful. If of greater severity, the measures mentioned above, such as glycer powder, infusion of senna pods, salines, massage, and others given on p. 275 may be used.

Failure to gain Weight is usually due to some gastro-intestinal disorder. In the absence of this, lack of progress points to the desirability of increasing the strength or the amount of the child's food.

We may now pass on to the treatment of the causes of these digestive disorders, dealing with the necessary alterations in the diet.

Excessive Volume of each Feed and Excess of Carbohydrate.—These have already been dealt with (pp. 33 and 38).

Fat-indigestion.—This is easily treated by the diminution of the amount of fat in the diet. It is not a common condition in infants fed according to the ordinary rules, but is frequent after an attack of diarrhoea. Where no fat can be tolerated a diet of whey, albumen-water, raw meat juice, and albumactin may be given temporarily (see Fig. 9, p. 44).

Curd-indigestion.—This is the most frequent cause of digestive disorders in bottle-fed children. The chief difficulty in rearing infants on cow's milk lies in the indigestibility of its curd as compared with that of human milk.

As has been mentioned, curd-indigestion is recognized by the character of the patient's motions. In addition to treating the symptoms of this disorder in the ways described, we have to consider how we can modify the feeds so as to render digestion more effectual.

We may endeavour to overcome curd-indigestion by further *division of the milk*. The weakest dilutions of milk that can be given, except temporarily, without risk of producing rickets, are, in proportions of milk to water, 2 to 1, 2 to 3, 3 to 1 at the ages of three, six, and nine months respectively. Free dilution of milk is therefore of chief value in the case of very young infants, and should not be allowed for any length of time without the addition of cream.

A more effectual method is that of *citration* (p. 36). This so profoundly modifies the curd of milk that we may usually escape the necessity for adding further diluent, and thus obviate the difficulties in connection with increasing the volume of the feed, or any deficiency in fat. We may even dispense with the diluent altogether in the case of infants over the age of six or eight weeks. A diet of citrated whole milk is particularly useful in wasted infants whose stomachs are intolerant of any large feed. Citration is so effectual, so cheap, clean, and easy, that it should always be tried first in a case of curd-indigestion. Full citration, 2 grains of citrate to each ounce of milk, should be practised until about the age of six months, when the amount of the salt may be gradually reduced.

Should citration fail—and we are now dealing with a comparatively uncommon case—we may turn to peptonized, *humanized*, *desiccated* or *condensed* milk. *Peptonized* milk (p. 40), is not very likely to succeed where citration has failed, unless peptonization is undertaken for twenty or thirty minutes, in which case the milk tastes very bitter. These are, however, a few cases in which the digestive tract is much atrophied, which do well on this. *Humanized* milk (p. 42) is a valuable preparation in cases of curd-indigestion. The method of making it is complicated and not well adapted to a poor or dirty home, and therefore *humanized* milk as best used is families that can afford to

buy it ready for use. Citrated lactinized milk can also be bought, but such is rarely necessary. *Desigated* milk (p. 48) is a good preparation for poorer families, although not suitable, unless supplied free, for the most poverty-stricken. It is easily mixed for administration, and fairly easily kept uncontaminated in the process. *Condensed* milk (p. 50) is fairly cheap and convenient in use, and may be of value as a temporary measure. It is not likely to suit many cases in which citation has been given a really proper trial.

Should curd-indigestion still be present, we may make temporary use of *milk* (p. 49), or—a much more valuable step—obtain the services of a *wet-nurse* (p. 22). Both these methods are, however, only applicable to the infants of the well-to-do.

More often it happens that we must fall back upon the use of a food free from curd-forming protein, of which the best is a *whoy* and cream mixture with raw sweet pure (p. 49). In the case of the very poor a very dilute solution of sweetened condensed milk may be used as a new approach to *whoy*. We must, however, recognize that such measures are for temporary use only, so that as soon as the infant's digestion has improved sufficiently, milk, preferably citrated, should be very gradually re-introduced into the dietary, by adding it in slowly increasing quantities to the *whoy*.

Sometimes the addition of some starch-free food may be beneficial towards the end of the first six months of life, in order to increase the nutritive value of the food without increasing the amount of casein in it. For this purpose *Mellin's Food* (p. 32) may be given in the proportion of not more than one heaped teaspoonful to every 3 oz. of the milk mixture.

Starch may be given as soon as the baby has cut one or two teeth, or between the ages of six and eight months. It is, however, worth bearing in mind that occasionally the administration of starch may be beneficial even before the sixth month of life.

General Rules.—I have endeavoured to give some idea of the plan of procedure which should be in our minds when we are confronted by a difficult case of infant-feeding, and it now only remains to emphasize some rules which, although well recognized, are of too great importance not to be repeated.

In the first place, changes in an infant's diet must not be made too frequently. There is a danger of nothing suiting because nothing is given a fair trial.

Secondly, all changes towards strengthening an infant's diet must be made very gradually. The change, for example, from citrated or peptonized milk to ordinary milk must be made in a number of small steps carefully watched. The same rule applies to the re-introduction of milk into a *whoy* and cream mixture, or to the change from bottle- to bottle-feeding. The more gradual the changes, the more successful shall we be in infant-feeding.

Thirdly, for an artificially fed baby, cow's milk is the best food, and every effort should be made to find some form of this which will suit the child. All other foods are, as a general rule, to be looked upon as temporary expedients only. Even a carefully prepared mixture of whey, cream, and raw meat juice is no exception to this rule. The more adept we are in modifying cow's milk for infant feeding, the less use we shall have for other methods of artificial feeding, and the more successful we shall be.

Lastly, as a rule, an infant's digestion begins to improve at about the age of six months, and if we can keep the child alive until that time, we may look for some diminution of our difficulties.

SECTION III.

CONSTITUTIONAL DISEASES.

I.—RICKETS.

"*The Rickets*" was first described by Gasson in 1690. As an alternative title he suggested the term "*Rachitis*," partly on account of its similarity to the English name, but also as emphasizing the affection of the spine (*kyph*) so frequently found in the disease. As it was first described in this country, it is known in Germany as "*the English disease*." Under the names of "*acute*" and "*fatal*" rickets were formerly described infantile scurvy and achondroplasia.

Etiology.—Without doubt, rickets is to be regarded as a dietary disease. While a deficiency in the diet of protein and possibly of salts may be contributing factors in its production, the most constant and potent cause is either an inadequate supply or a defective assimilation of fat.

Normal human milk should contain 5.5 per cent of fat, and to this standard an infant's diet should comply. Deficiency in the supply of fat is most commonly due to one of three causes—over-prolonged breast-feeding, feeding on condensed milk or proprietary foods, or the use of too freely diluted cow's milk. Breast milk, when poor in quality, is usually deficient in fat, and such a defect, while possible at any time during lactation, becomes almost inevitable after the ninth month. In condensed milk given diluted to 1:10, as is often done, fat may only be present to the amount of 0.5 per cent (Still), and many of the worst cases of rickets arise from the use of such milk and of the proprietary foods. Cow's milk, as sold in London, shows on an average 3.66 per cent of fat (Maloney), and where it has been given diluted and without the addition of cream, over a long period, rickets may result. Where water is added to milk in a greater proportion than 1 to 1 at three months, 1 to 2 at six months, and 1 to 3 at nine months, there is a danger of causing rickets.

On the other hand, fat, although given in sufficient quantities, may not be assimilated. Immaturity, dyspepsia, tuberculosis and syphilis, are the chief causes of such a condition. The relationship between rickets and syphilis is to be explained in this way. Similarly, the premature administration of starchy foods, or an excess of sugar in the

diet, may predispose to rickets by setting up digestive disorders which prevent the absorption of the proper amount of fat.

The children of the poor are more commonly rickety than those of the well-to-do, owing to the more frequent use amongst them of the faulty methods of feeding which have been mentioned. Some authors invoke defective hygiene, over-crowding, absence of fresh air and sunlight, as direct causes of rickets; but it is more probable that these are to be regarded chiefly as mere associations of poverty, although they may tend to produce malassimilation of food.

Age.—It is doubtful if rickets is ever truly congenital; certainly it is very rarely met with before the third month of life. By the sixth month it is not uncommonly recognizable; but its most pronounced



Fig. 161.—RICKETS: SWELLING DEVELOPED AT BASE OF FINGER, CLAVICLE, AND LILLO BONES. Note Deformity of Finger of Right Hand. (Dun. 1910.)

features do not appear usually until the second year. The active stage of the disease ceases during the third and fourth years, except in those rare cases known as "late rickets" (p. 72).

Symptomatology.—Rickets is not a disease of the osseous system alone. Although the bony changes occur early and render the diagnosis easy, symptoms may arise from the respiratory, alimentary, muscular or nervous systems, and these may dominate the clinical picture.

In the worst cases, severe wasting may be present, but in many the child is fat, often too fat, pale and flabby. The temperature is not raised unless some complication be present, although there is apparently some sensation of heat, if we judge by the frequency with which the

bed-clothes are kicked aside at night by the patient. Sweating of the head, often very profuse, may occur during sleep.

Bony Changes.—These, as a rule, are first seen in the beading of the ribs. Rounded eminences develop at the costochondral junctions, those of the sixth ribs being usually the first affected. A row of these beads forms the "rickety rosary." Similar changes may occur internally at these junctions. The chest wall, which is usually stiff and yielding, easily undergoes changes in shape. There is a tendency for the lower ribs to become everted, and for a transverse groove to appear, stretching from the costliest cartilage to the posterior axillary line (Harrison's suture). In more severe cases, the lateral aspects of



FIG. 11.—(ENLARGED) POSTERIOR ASPECT OF DISTENDED CERVIX.

the chest may become sunken by the development of longitudinal grooves, the costal cartilages being protruded forwards and the ribs, and possibly the clavicles also, being fractured. These changes, which are seen in Figs. 10 and 11, are associated with more or less collapse of the lungs. While rickets will predispose towards any type of deformity of the chest, it will be seen that the purely rachitic thorax, with its lateral depressions, differs from the true "pigeon-chest." In the latter, the ribs are strengthened and meet at an angle at the sternum, so that in transverse section the outline of the thorax is triangular (Figs. 12 and 13). In both, the capacity of the thorax is diminished.

The head shows many important signs. The anterior fontanelle, which should measure an inch or less at the twelfth month, and be

closed by the eighteenth month, remains unduly large, and closes late, owing to defective bone-formation. The edges of the bones enclosing the fontanelle are often slightly thickened. A yielding state of the membranous bones of the cranium, enabling them to be indented easily by the pressure of the finger, may be present. It is termed *craniotabes*, and exists in the two forms, a diffuse or a localized yielding. In the former there are diffuse areas of yielding bone close to the lambdoid and coronal sutures. The condition is most commonly found in the posterior parts of the parietal bones. Such a state, which can hardly be regarded as pathological during the first few months of life, is very commonly present in rachitic infants. The localized thinning is less common but of more significance. To this it is best to limit the term



Fig. 11.—Rickety head.

craniotabes. In this form there are small areas, often usually near the lambdoid suture, where the bone has become absorbed.

The common rachitic head is the large, square or box-shaped head. All its surfaces are flattened, and in profile only the tip of the nose projects beyond the plane of the face. The forehead is high and broad; it is sometimes well termed the "pseudo-intellectual forehead." Less commonly, the rickety head is elongated from before backwards, while compressed laterally (Fig. 11). In addition, bossing of the skull may be present, in which case rounded symmetrical eminences are developed on the frontal and parietal bones. The four bones are separated from each other by a cruciform depression, so that the condition is sometimes spoken of as the "bat-eaten head" (Fig. 13). Formerly, both head-bossing and *craniotabes* were regarded as evidence of inherited syphilis rather than of rickets; but it seems certain that

both these conditions may occur from rickets alone, although in the worst cases the syphilitic taint is also present.

Delayed dentition is a very constant sign of rickets. Frequently the first teeth do not appear until after the twelfth month. Their eruption is likely to be associated with various nervous disorders in rickety children. When cut, the teeth tend to be frail and notched, and as their enamel covering is poorly developed, they are prone to early decay. Dentition, should it have begun, may become suspended with the onset of the disease.

The spine frequently shows the well-known curvature. It is usually more rounded than the angular deformity of tuberculous caries, and it



FIG. 13.—RICKETS: "HUMP-BACKED" HEAD.

is easily distinguished from this more serious disease by the absence of rigidity, the spinal column becoming straight on extension, provided the child is quiet and not resisting (Fig. 14).

The long bones show many changes. The earliest is the enlargement of the lower radial epiphyses, those of the other bones being usually affected later. As the result of crawling, walking, or even of being carried, the child may develop various curvatures, usually more marked in the bones of the lower extremities than in those of the arms. The commonest deformity is a sharp lateral curve in the lower part of the tibia, with which may be associated a forward bending of the shaft of

the bone (Fig. 13). Such may develop before the child can walk, from the cross-legged position adopted in sitting. *Genu valgum* is



FIG. 14.—RICKETS: THE "CAPE NECK."

This posture may lead to curving of the spine if the arms.

very common. Less often there occurs the condition known as *coxa vara*, in which bending occurs in the neck of the femur, so that the head of the bone comes to lie as low as the great trochanter, or even at a lower level. The pelvis may be deformed in various ways, leading to a diminution of its capacity. The bones of the arms may become curved from the child putting its weight upon them when crawling or sitting on the floor (Fig. 14). Fractures are common. There may be considerable stunting of the growth of the bones of the limbs in severe cases of rickets.

Muscular System.—Rickets is a common cause of a child going "off his legs." The muscles become much weakened, so that the patient is no longer able to support his weight upon them, although still able to move them freely. At an earlier age, rickets is the most



FIG. 15.—RICKETS.

A common deformity of the legs.

frequent cause of delay in the attainment of the power of walking. In most cases there is a considerable degree of muscular flabbiness which, with the laxity of the ligaments that is often present, renders



FIG. 10.—RICKETY DEFORMITIES.

the limbs very hypotonic (rickety hypotonia) and permits of excessive movement at the joints (Figs. 10 and 11). In these children, the toes can be easily made to touch the ear, or the dorsum of the foot be brought in contact with the front of the leg. These changes may overshadow the bony signs, as in cases which are sometimes termed "orthotic rickets." In rickety children the recti abdominis muscles are often greatly separated at their upper parts, owing to the expansion of the costal arch and the enlargement of the abdomen. This frequently leads to the development of a ventral hernia. The atonic state of the abdominal wall and of the muscle of the intestine are two of the factors in the production of the rickety "pot belly."

Respiratory System.—Nasal laryngeal and bronchial catarrhs are common in the subjects of rickets and are apt to recur repeatedly unless the underlying condition be recognized and treated. Pulmonary collapse is often induced owing to the yielding nature of the chest-wall. Broncho-pneumonia commonly supervenes in an attack of bronchitis, and the softness of the chest-wall and the abdominal distention tend to cause collapse of the lung and impeded action of the diaphragm, so rendering the dyspnea worse, and the course of the disease less favorable. Laryngeal spasm is dealt with later.



FIG. 11.—RICKETY DEFORMITIES.

Gastro-intestinal System.—Here, too, there is a tendency to catarrhal conditions. Attacks of diarrhoea are common in rickety children. Irregularity of the bowels and a distended distention of the intestine are very frequent. The well-known "pot-belly" of rickets is produced by many factors; the smallness of the thorax and pelvis, the eversion of the lower ribs, the atony of the abdominal and intestinal muscles, the distention of the bowels, and the enlargement of the liver and spleen, may all conduce to the production of the large protruding abdomen (Fig. 10).

The term "catarrhal rickets" is sometimes given to that important group of cases in which catarrh of the respiratory and alimentary systems are conspicuous, while the bony changes are slight.

Both the liver and spleen may be slightly enlarged. Owing to the eversion of the lower ribs and other causes, these organs may appear clinically to be more enlarged than is actually the case, and may be palpable an inch or two below the costal margin.

Circulatory System.—Anæmia is often present, and may be severe. The blood shows only chlorotic changes. This is therefore one of the conditions of anæmia with a (clinically) enlarged spleen.

Nervous System.—The nervous system is unstable in rickets. Convulsions associated with dentition, gastro-intestinal derangement and other conditions, are more common in rachitic than in non-rachitic children. Laryngismus stridulus, facial irascibility, and tetany are practically confined to rickety infants; while the condition known as head-rolling and nystagmus is most commonly seen in the subjects of this disease. These disorders are described under "Functional Nervous Disorders."

The children are often very restless, especially when asleep, and during the waking hours riding movements of the head upon the pillow may be so constant as to cause the hair on the back of the head to be worn away. The patients often show much irritability, so that it is difficult to say whether the limbs are tender or not; but should there be definite tenderness present there is a scurvy element in the condition. Rickets alone does not produce tenderness. If the tone of the muscles be good the deep reflexes are usually lively.

The association between rickets and catarrh has long been known.

Rickets has been said to be a cause of mental precocity by some authors and of intellectual deficiency by others. From this one may conclude, what seems to be the fact, that the disease leaves no direct trace upon the mental life of the patient.

Morbid Anatomy.—The changes in the bones are summed up in Sir William Janssen's words as "extensive preparation for ossification and imperfect performance of the process." In intracartilaginous ossification, the predominant zone is greatly increased in thickness, and presents to the zone of calcification a very irregular instead of a regular edge. Thus the two areas become mixed, small islets of

calcification lying in the cartilage, and irregular processes of cartilage extending into the area of calcification. Microscopically, the cartilage cells are seen to be increased in number and to be laid down irregularly instead of in the usual parallel columns. Throughout this abnormal area there is increased vascularity and a too rapid absorption of the cartilagenous tissue takes place, leading to weakening of the bone. The bone formation under the periosteum is similarly imperfect. The periosteum is thickened, and the proliferating layer under it shows increased vascularity and deficient calcification, so that the bone laid down here is abnormally soft. In the cranial bones, particularly, an excessive amount of soft spongy bone may be formed, and give rise to the bowing of the skull. On analysis the lime-salts are found to be greatly reduced in amount, even to less than one-half of the normal. This cannot as yet be satisfactorily explained by any of the many theories which have been put forward.

The muscles may show excess of fat between their fibres, or even fatty degeneration of the cells themselves. The spleen shows hyperplasia of the splenic pulp, and in some cases a diffuse thrombosis. The actual increase in the size of the organ is never more than slight. The liver is frequently slightly enlarged and fatty, particularly where there has been much diarrhoea, and may show some increase of fibrous tissue. The respiratory system, the alimentary tract, and the brain, show no abnormalities peculiar to rickets.

Diagnosis.—A case which is essentially one of rickets may be brought to the physician for many reasons other than that of bending of the bones. Bronchitis, diarrhoea, anaemia, convulsions, tetany, inability to walk, and enlargement of the abdomen may be cited as common symptoms which are the cause of the child's visit to the doctor. In such conditions the underlying rickets is often all-important; and inasmuch as the bony signs are not necessarily well-marked, it is useful to bear in mind which of them are of the most value in the diagnosis of difficult cases. Of all the osseous changes in rickets, the most constant are bowing of the ribs, retarded dentition, and delayed closure of the anterior fontanelle. Where the weakness is very marked, rickets may be confused with a nervous condition such as acute polyomyelitis or post-diphtheritic paralysis. From the former it is distinguished by the fact that it is a weakness of gradual, and not a paralysis of sudden, onset; and from the latter by the absence of involvement of the cranial nerves and the retention of the deep reflexes.

The globular head of hydrocephalus can usually be easily distinguished from the enlarged rickety head with its flattened surfaces. Occasionally, however, the diagnosis can only be made by watching the growth of the size of the skull, and for the onset of signs of raised intracranial pressure in the hydrocephalic cases. The spinal curvature of rickets has already been discussed (p. 66).

Prognosis.—The disease is very amenable to treatment, and may always be brought to a standstill, although its results may be permanent. The active stage of the disease ceases of itself during the third year of life. Recrudescences after the fourth year are very rare, and form the majority of the cases known as "late rickets" (p. 72). In an indirect way, rickets is responsible for a large mortality; respiratory diseases, diarrhoea, and convulsions being the most common actual causes of death. *Laryngismus stridulus* is very occasionally fatal.

Treatment.—Sufficient has been said on the subject of the prevention of rickets in dealing with the cause of the disease.

The curative treatment includes dietetic, medicinal, and general measures, the treatment of deformities and of special symptoms.

The articles of diet which are rich in fat and suitable for infants, are milk, cream, butter, bacon-fat and yolk-of-egg. The last should be from a lightly boiled egg, the whole of a yolk being given daily to a child of a year old.

Amongst drugs, cod-liver oil takes the first place, owing to the ease with which it is assimilated. It may be given plain, in doses of fifteen minims for an infant of six months, to half a drachm for a child of two years. It is usually better taken in the form of an emulsion. The emulsions prepared by the best firms are thoroughly reliable. For hospital patients, such a cod-liver oil mixture as is in use at the Paddington Green Children's Hospital is very satisfactory. —

R. Oil. cod-liv.	℞iix	Tragacanth.	℥ss
Glycerin.	℞vj	Aq. destil.	(to 5)
℞iij calc. sacch.	℞iis		

Some children will take a sweeter mixture more readily, as, for example, one containing 20 minims each of oil, lime-water, and glycerin. By the addition of malt extract, cod-liver oil is made much more palatable. If the administration of iron is indicated, 15 minims of the compound syrup of the phosphate of iron may be added to a drachm of the mixture given above. A stronger preparation is the mixture of equal parts of oil and steel wine. "Paroleum" is a good preparation.

Cod-liver oil is best taken directly after food, and should not be given if the digestive tract is in any way out of order. Other oils, such as that of cotton-seed, are more apt to upset the digestive system than is cod-liver oil.

The use of phosphorus and the phosphates has been advocated by some authors, but their curative action upon rickets is very doubtful, and they are no longer used by English practitioners for this disease.

Although it is impossible to regard lack of fresh air and sunshine as in themselves direct causes of rickets, it is certain that favourable climatic conditions are of great help in the cure of the disease. For

this reason, where possible, the child should be removed to some sunny, breezy place, preferably on the sea coast.

In order to prevent the onset of deformities, it is as a rule necessary to enforce rest. This is most conveniently done by applying to the legs light wooden splints which are sufficiently long to project beyond the feet. These, which should be removed at night, prevent the child from standing on its feet. They should be worn for several months, especially if the patient be fat and heavy. Where there is much spinal weakness, the child should be kept lying down as much as possible. If necessary, this position also may be enforced by the use of long splints.

For the atony of the muscles, gentle massage and warm baths with tepid soaping are of service. The child should be encouraged to kick its legs about when the splints are removed at night.

The catarrhal complications must be guarded against. The methods of administering iron for the anemia have already been given. Tenderness of the limbs is quickly amenable to treatment by antiseptic liniments.

The nervous symptoms will require antispasmodic measures (baths, etc.) and sedatives, such as bromide, digitalis, or opium.

After the cessation of the active stage of the disease, surgical treatment may be necessary for the correction of deformities.

CONGENITAL RICKETS.

It is questionable if rickets is ever truly congenital. Such cases as have been described as instances of congenital rickets have shown a marked tendency for spontaneous fractures to occur in the bones, and thus seem to belong to the group of cases now called "osteogenesis imperfecta."

Fetal rickets was the name under which achondroplasia was formerly described.

LATE RICKETS.

Under the title of late or delayed rickets, a group of rare cases is described, in which rickets either develops for the first time, or reappears, after the age of four years. The majority of the cases belong to the latter group, and would be more properly classed as recrudescent rickets. Most commonly, late rickets appears between the ages of nine and fourteen. No dietetic cause can always be assigned; sometimes an illness precedes the onset of the disease. The limbs show the same changes as they do in the infantile disease, but are usually the seat of slight pain. The skull, however, remains unchanged, and there is little or no tendency to respiratory complications.

Late rickets has to be distinguished on the one hand from a simple bending of the long bones which may occur from too rapid growth, and on the other from osteomalacia of the adult type. The latter disease is extremely rare before the age of puberty, but has been

recorded as occurring as early as the fifth year. It may be suspected where the degree of bone-softening is out of all proportion to the other obvious signs of rickets.

The *Fendlicur* which appears to be most useful is that which is of benefit in infantile cases. Severe deformities may arise which will require surgical treatment when the active stage of the disease comes to its end.

II—INFANTILE SCURVY

INFANTILE SCURVY was first recognized by Gibson in his original description of rickets, but later it became regarded as an acute form of rickets until 1878. In that year, and again in 1882, Chevalé showed that infantile scurvy was identical with the scurvy of adults, and was a disease distinct from rickets. In 1883 Sir Thomas Barlow published his well-known account of scurvy, and by Continental authors the condition is often called by his name. The terms acute rickets and scurvy-rickets should no longer be used, for it is now well recognized that scurvy and rickets are two separate diseases, both of which are due to the same dietary, although for different reasons.

Etiology.—Although the theory is beginning to be much criticized, the most commonly held as to the origin of scurvy is that it is caused by a diet which, by sterilization, has been deprived of its "fresh element." It will be seen that this statement contains three points which are quite distinct and separate: first, that the disease is due to a food which has been sterilized; second, that it is due to the food because it has been sterilized; and third, that it is due to the destruction by sterilization of the "fresh element" in the food. It may be pointed out that the third proposition does not of necessity follow from the first two, and that this theory disregards the chemical composition of the food, and any possible alteration in it as the result of sterilization.

There are numerous difficulties in the way of accepting this theory in its entirety. We do not know of what this "fresh element" in the food consists; and further, it is extremely difficult to imagine of what kind of substance it could consist. What could be the nature of a "fresh element" of which much is present in some fresh foods, such as fruit-juices and little in others, such as fresh milk, or that is harmed by pasteurization and destroyed by prolonged sterilization? Clearly it cannot be an enzyme, for pasteurization would effectively destroy it. Again if the "fresh element" is the all-important factor in the prevention of scurvy, how is it that cases arise occasionally in children who are entirely breast-fed (Holt)—and how is it that scurvy can be cured just as well by the administration of boiled fruit-juices,

ness of these which have not been boiled? We must conclude, therefore, that we cannot invoke the "freshness" of a substance as being in itself therapeutically active. Such an argument is comparable to explaining the value of chlorophyll-containing vegetables in the treatment of anemia, by saying that it is due to their "greenness."

Nor can scurvy be regarded as due to some injurious content of the diet, for the disease may be cured by the simple addition of fruit-juices to the diet upon which the symptoms have arisen. In the feeding, that is to say, there has been an error of omission rather than of commission.

When we turn to the chemical aspect of the subject, we seem to find an elucidation of the problem. Sir A. E. Wright has shown that in scurvy there is a diminution of the alkalinity of the blood, and he regards this as the essential factor of the disease. If the diet is deficient in alkaline salts, scurvy may be produced, and it may be cured by the administration of these salts in which vegetables and fruits are very rich. More has been done to prove this view, which was first suggested by Ralfe, in adult than in infantile scurvy; but several facts are of interest in connection with the latter. Where a cereal food, which when burnt yields an acid ash, is being given, the scurvy will be cured merely by its withdrawal (Hutchinson). Scurvy can be cured, not only as has been mentioned, by the administration of boiled fruit-juices, but by the giving of sodium citrate as a drug. When under treatment, the patient's blood becomes more alkaline and the urine becomes alkaline in reaction. The question of the sterilization of milk is of great importance, and its possible danger can be explained by this theory of the causation of scurvy. Fresh milk contains citrate of lime in its amorphous and soluble form, but when the milk is boiled this salt is changed to its crystalline form, which is less soluble, and so tends to separate out and sink to the bottom of the fluid. It can be easily understood, therefore, how boiling and, still more, prolonged sterilization, may tend to produce scurvy. If however the milk is sterilized in small bottles, each containing only enough for one feed, the danger of a lack of the alkaline salt in the milk taken by the child is avoided. This has been shown clinically by Bodin in the feeding of many hundreds of infants in whom no case of scurvy arose. He used unboiled milk of good quality, which had been sterilized for forty minutes.

Some recent feeding experiments by Hôbl are strongly opposed to this chemical theory of the causation of scurvy. The question cannot therefore be regarded as settled at present, although the balance of evidence appears in favour of Wright's view.

Leaving these considerations, and approaching the etiology of scurvy from its clinical side, the vast majority of the cases are due to the use of proprietary foods; but the disease may arise from sterilized milk, less often from pasteurized milk, and very occasionally from fresh milk, even breast-milk. It tends to occur in the children of the

rich rather than those of the poor. The subjects of scurvy are nearly always fat babies with pretty pink-and-white complexions.

The age-incidence of scurvy is well worth remembering. The disease arises with great regularity between the sixth and twelfth months of life. Most of the cases occur just when the first teeth are appearing, that is during the sixth, seventh and eighth months.

Symptomatology.—*Swelling* in an infant who is well-nourished but showing the earliest signs of rickets, scurvy produces its symptoms by the hemorrhages which take place. The child may be brought to



Fig. 15.—Scurvy. Hemorrhage into Gums before Eruption of New Teeth.

the doctor for tenderness or swelling of the limbs, or for what is thought to be pusulitis, or even meningitis. With a hemorrhage the temperature rises to 100° or 102° , and generally remains slightly raised while the absorption of the blood proceeds. Hemorrhages may occur anywhere, but the most characteristic signs and symptoms are those here described.

Generalized tenderness is perhaps the earliest sign of all, and is not uncommonly seen in rickety children who show no other evidence of scurvy. It is, however, to be regarded and treated as a scorbutic sign, and not as one of rickets.

If the teeth are in, or on the point of eruption, hæmorrhage into the gums is the most constant sign of the disease. The gums become spongy and purple, and may be so swollen as to project from the mouth. The gum affection of scurvy shows two points of great clinical importance: first, it is only present if the teeth are erupted or on the point of being out (Fig. 18); secondly, where the teeth are through, it is practically always present in a case of scurvy. Thus, in a child with teeth erupted and normal gums, a diagnosis of scurvy would almost certainly be wrong.

Hæmaturia is very commonly present; indeed, microscopical evidence of such is said to be constant in scurvy. In some cases the



FIG. 18.—SCURVY. SWELLING OF RIGHT THIGH DUE TO SUBPERIOSTEAL HÆMORRHAGE.

hæmaturia is the most marked sign, and occasionally is the only symptom of the disease. It is well to remember that hæmaturia occurring at the scurvy age is usually due to scurvy.

Swelling of the limbs due to subperiosteal hæmorrhages is the most common cause of the child being brought to the doctor. Such hæmorrhages occur most frequently in the lower end of the femur or in the tibia, the bones of the upper extremities being less often affected. The skin over the swelling is tense and shiny, but neither hot nor discoloured. The lesions are often multiple, and may be symmetrical.

The swelling is exquisitely tender, and any movement or handling causes the most intense pain. It is characteristic of the subjects of this disease, that they scream lustily when approached or when the bedclothes are raised. This screaming often gives rise to the false diagnosis of meningitis; but it should cause no such mistake, for an infant with meningitis does not scream when approached, being in too drowsy a condition to take notice of what is going on round it. The extreme painfulness of the hemorrhages causes the affected limb to be held absolutely at rest, so that paralysis may be suspected. The swelling, which results from the extensive bleeding underneath the



Fig. 10.—SCURVY. HEMORRHAGE UNDER LEFT EYE.

periosteum of the bone, extends from just above the joint up the shaft of the bone (Fig. 15). Fracture at the epiphyseal line may be caused by hemorrhage occurring there, while occasionally blood is effused into the joint itself. From bleeding into the costochondral articulations, there may be flattening and sinking in of the sternum and costal cartilages.

The eyeballs may become swollen and discoloured from bleeding into their soft tissues (Figs. 20 and 21); while displacement of the eye forwards or downwards may occur from bleeding beneath the pericranium of the root of the orbit. Such proptosis arises suddenly, and

although it is not very common, it may be the most conspicuous early sign of the disease.

The skin and mucous surfaces may show purpuric spots. Hemorrhage may occur into the bites or vaccination-puncta. In neglected cases, the whole surface of the body becomes extraordinarily itched.

Less commonly, bleeding may take place into the gastro-intestinal tract, while rarely, the brain or lungs may be the seat of hemorrhage. In the last situation it may be suspected where there is much dyspnea.

Morbid Anatomy.—The most definite changes are in the bones. The periosteum over the hemorrhage is thickened and extremely vascular, and the blood-clot shows varying degrees of organization. The muscles in the neighbourhood may

be infiltrated with blood. In very chronic cases the separated periosteum may go on forming bone, so that a layer of poorly developed bone is found on the surface of the hemorrhage (Fig. 22). The bone itself is softened and the marrow highly vascular. The changes due to rickets may also be present. Separation of the epiphyses or fracture near the epiphyseal line of the bone may be found.

The blood shows a diminished alkalinity. There may be a chronic change present unaccompanied by any definite leucocytosis. The anemia is not entirely due to hemorrhage.

Diagnosis.—There is seldom any difficulty in recognizing scurvy if its possibility be remembered. The age of the child, the characteristic tenderness, the condition of the gums, and the changes in the urine, as a rule make the diagnosis clear. The pain in the limbs may suggest



FIG. 21.—SCURVY. HEMORRHAGIC CIRCLES. CIRCLES ON LEFT EYE.



FIG. 22.—SCURVY. SEPARATED BLOOD-CLOT AND NEW BONE FORMATION.

dermatitis), but this mistake should never be made, for rheumatism does not occur at the scurvy age. The screaming sometimes leads to a diagnosis of meningitis, but this sign is by no means sufficient to lead to such a conclusion. Infantile paralysis may show considerable hyperesthesia, and the immobile tender limb of scurvy may be thought to be due to acute poliomyelitis; in the latter, however, there is no swelling of the limb, while the other signs of scurvy will be absent.

Two conditions are with much greater difficulty distinguished from scurvy: namely, syphilitic epiphysitis, and acute osteomyelitis. In the former of these, the limb is very tender and is kept unmoved as in scurvy; but it occurs in younger children (practically always under three months), showing signs of inherited syphilis, while the swelling of the limb is more localized to the epiphysis. To determine whether the swelling in the limb is a subperiosteal hemorrhage of scurvy or a subperiosteal abscess from deep-seated suppuration may be most difficult. The extent of the fever is of importance; but a low temperature does not absolutely exclude suppurative changes, though they are not likely to be present under such circumstances. A leucocytic count may be of service. The other signs of scurvy will of course settle the matter.

Sarcoma of the skull may simulate the orbital hemorrhages. Scurvy must be remembered as a common cause of hematemesis in infants of six to twelve months of age.

In any case of suspected scurvy, the therapeutic test may be of great use—three days' treatment on antiscorbutic food should bring about a very definite improvement in the tenderness.

Prognosis.—No disease is more immediately under control than is scurvy. If recognized and treated, the symptoms begin to show noticeable improvement within two or three days, the first to become modified being the tenderness. Occasionally, during the first few days of treatment, although the pain is lessening, a fresh hemorrhage may occur either spontaneously or as a result of mistreatment, this is, however, exceptional. The hemorrhages take some time to absorb; the fractures to unite. Death but very rarely occurs; diarrhea, possibly hemorrhage into the lungs, or broncho-pneumonia, may cause death.

Treatment.—All handling of the child should be avoided as far as possible. If it must be moved it should be carried on a pillow. The affected limbs may be surrounded loosely by wool, and the bed clothes should be raised off them by a cradle. The child's clothing should be arranged so that it may be removed with as little movement of the patient as possible. The mouth may require special care.

The alkaline salts requisite may be supplied by the administration of orange juice, grape juice, or "potato cream" (made by rubbing

up the solid part under the skin of a baked potato with milk). These may be given in fractional doses four- or six-hourly. Fine meat juice (up to half an ounce daily) is of service; but both it and potato cream tend to produce diarrhoea, so that they must be given carefully, for diarrhoea is apt to give rise to a tetrademolysis of the haemoglobin, and may of itself be dangerous. The citrate of sodium exerts a cathartic influence. During convalescence cod-liver oil and iron may be of service.

III—DIABETES MELLITUS.

The two most important points about this disease as it occurs in children are, first, its comparative rarity; and second, the severity of its clinical course. Out of 8,000 post mortems made at the Hospital for Sick Children, Great Ormond Street, London, only ten were on subjects of this disease. It has been recorded in new-born children; but it must be remembered that lactaria is not uncommon in nurslings.

The disease runs a rapid and unfavourable course: death may occur within six weeks, and usually takes place within a year.

The pathological problems are the same as in the severe form of the disease in adults, and the treatment differs in no way. Coma always tends to be threatening in these children, as is shown by the presence of diacetic acid in the urine by the ferric-chloride test. The carbohydrate in the diet must therefore be reduced extremely cautiously and gradually; constipation must be avoided, and alkalis given in an endeavour to counteract the acid intoxication of the blood.

Diabetes is the least common cause of acetonuria in children.

IV—ACIDOSIS AND ACID INTOXICATION.

(Acetonuria.)

This subject is one of great importance and of no little difficulty in connection with the diseases of children.

A distinction is made between the two terms *acidoses* and *acid intoxication*. By the former is meant the presence of abnormal amounts of certain organic acids in the blood and urine. Acid intoxication, on the other hand, refers to the toxic symptoms which may be produced by these circulating acids. Acidoses may, however, exist without any symptoms of acid intoxication.

Much attention has been paid to the subject of acetonuria of life, and general agreement has been reached on certain points which may

be briefly summarized. It seems clearly proved that the acetone group of substances (β -oxybutyric acid, diacetic acid, and acetone) are the products of the breaking down of fats and of fats only. They are, therefore, likely to be increased in amount where the diet is rich in fat. But on the other hand, their production is closely connected with the amount of carbohydrate ingested, for the reason that normally, carbohydrate metabolism tends to spare fat metabolism. From this it follows that the withdrawal of carbohydrate from the diet will tend to increase the breaking down of the fats of the body, and thus cause an increased formation of the acetone group of substances. This, it will be obvious, has a most important bearing on the dieting of severe cases of diabetes, in which acid intoxication is threatening, and also upon the treatment of other conditions of the same nature. Starvation, usually from vomiting and diarrhoea, causes increased fat metabolism, and consequently over-production of the acetone bodies. Constipation is also a potent cause, as is well recognized in the prevention of diabetic coma.

The toxic properties of the acetone group of substances do not lie in the presence of the acetone itself, but are due to the β -oxybutyric acid with which it is associated. Nevertheless, acetone is of great clinical importance, for it is readily recognizable by its odour in the breath and by chemical reactions in the freshly passed urine, while its amount varies directly with that of β -oxybutyric acid. Further, the symptoms are due to the fact that acid is circulating in the blood, and not to the particular kind of acid present. It is the acidity, that is to say, of the circulating acid, rather than the actual nature of that acid, that is the cause of the symptoms. This fact is of great clinical value, for it follows that in a case in which it is difficult to determine the amount of acid intoxication present, the administration of alkalis will help towards a definite conclusion. For if alkalis are given in sufficient amount to render the urine alkaline, the symptoms of acid intoxication must disappear; and if any symptoms remain, they cannot be due to this, but to some underlying condition. It may, for instance, be very difficult to decide if a case is one of tuberculous meningitis, with acetonaemia secondary to the vomiting and constipation present, or if it is one of cyclic vomiting (p. 83). If the urine be rendered alkaline by the administration of sodium bicarbonate, the symptoms due to the acid intoxication cease, and the diagnosis can then be readily made. In the worst cases of acid intoxication, so great is the production of the acid that the urine cannot be rendered alkaline, and death ensues.

Symptomatology.—With the onset of acid intoxication, the child becomes drowsy and passes into a restless, semi-delirious state, which in severe cases develops into actual coma, in which death may occur. Combined with the restlessness, is the well-known symptom of "air-hunger" with its characteristic gasping respirations. The breath

is laden with the sweetish odour of acetone, which may be appreciable at a distance from the patient's mouth. The urine contains much acetone (for tests see p. 85). Vomiting is a very constant symptom, and in bad cases is almost continuous. There may be diarrhoea, but more often the bowels are constipated. Jaundice may develop. The gastro-intestinal disturbances are of great importance, for it will be noted that they figure both as causes and symptoms of acid intoxication, and it is in the vicious circle thus set up that the danger of the condition arises.

Clinical Groups.—In one group we may put those instances of acid intoxication which are due to the gastro-intestinal disorders secondary to some easily recognizable condition. Thus, from pneumonia, acute infantile diarrhoea, tuberculous meningitis, intestinal parasites, and other causes of vomiting and diarrhoea or constipation, acid intoxication may occur owing to the condition of starvation obtaining for the time. Here the symptoms of acid intoxication are superimposed upon those of the underlying disease, and are rarely of themselves serious. The prognosis, for instance, in pneumonia does not appear to be much affected by the temporary acid intoxication. In this group of cases the symptoms can, as a rule, be quickly relieved by the administration of alkalis.

In a second group we may place those cases in which the symptoms of acid intoxication may be extremely severe and may cause death. In this class we have to mention (1) *Diabetic coma*; (2) *Post-anæsthetic acetonaemia*; (3) *The recurrent or cyclic vomiting of children*; and (4) *Salicylic acid poisoning*.

1. **Diabetic Coma.**—Mention has already been made of the great tendency to fatal coma in children with diabetes, and it is not necessary to refer further to this condition here.

2. **Post-anæsthetic Acetonaemia.**—This condition is sometimes termed "delayed chloroform poisoning", but this is to some extent a misleading name, as the acid intoxication may follow the administration of any anæsthetic. Chloroform, it is true, has been used in the majority of cases, but instances have followed after anæsthesia induced by ether, ethyl chloride, and even nitrous oxide (Langmead).

The cases usually arise in fat children, and probably for this reason often follow operations which are either slight in character, such as on tuberculous glands in the neck, or for the alleviation of some very acute condition such as acute appendicitis. The symptoms may appear within a few hours of the anæsthetic, or may be delayed for as long as three days after its administration. The temperature then becomes raised, even to 104° and all the characteristic symptoms already detailed develop. Death is unfortunately far from uncommon. Although it is difficult to say to what extent the ordinary post-anæsthetic symptoms are due to acid intoxication, it is certain that with the severe symptoms of at-burger and coma, death is very

likely to occur. In the fatal cases the liver invariably shows fatty degeneration, and in many the organ is composed almost entirely of fat, and is of a bright yellow colour. Such a liver is very characteristic of this condition and of the cyclical vomiting of children.

The origin of these cases is obscure; but it may be supposed that they occur only in children who are on the verge of acid intoxication, and in whom the circumstances of the operation tend to turn the balance, as it were, against the child. Thus it arises in fat children. The disease for which the operation is undertaken may have been treated by means of large doses of cod-liver oil, or may have been associated with vomiting and other gastrointestinal disturbances. In the preparation for the anæsthetic, the child may have been solidly-purged, and will almost certainly have been starved for some hours. After the anæsthetic, or even during its administration, some vomiting may occur. All anæsthetics, but especially chloroform, tend to inhibit the oxidation of fat during their exhibition. Such factors as these may be supposed to determine the onset of acid intoxication in a child already predisposed to that condition.

Treatment.—The prophylactic treatment is of importance. The urine should be examined for acetone, and if this be present in excess the operation should be postponed. All excess of fat in the diet should be avoided for some days preceding the anæsthetic, and particularly should the withdrawal of carbohydrates be avoided. During the hours preceding the anæsthetic, when food cannot well be given by the mouth, trachæta of glucose may be given. For two or three days before the operation, where possible, alkalis should be administered in full doses, so that the urine is made alkaline. Where such treatment has been carried out, according to Denker, the ordinary post-anæsthetic symptoms are much lessened.

Where symptoms have arisen, a purge may be given, the stomach washed out with alkaline lotion if the vomiting is severe, and alkalis and carbohydrates given in large quantities, by the mouth, per rectum or subcutaneously. Opium is of little service in checking the vomiting. As has been stated in the worst cases the urine remains acid and death ensues.

5. *Cyclical, Periodic or Recurrent Vomiting.*—Some children, usually of a neurotic stock, are subject to periodically recurring attacks of vomiting, with constipation or diarrhoea. These, which are usually regarded as "bilious attacks," tend to occur at intervals of a few weeks or months. Usually very slight symptoms of acid intoxication are present; but these in any attack may become severe, often very suddenly. More than one case may occur in a family. Jaundice may develop and does not necessarily forestall death. A fatal case is, indeed, very uncommon; but, should death occur, the bill-coloured liver, such as is found in post-anæsthetic acetonaemia, is found post mortem. What brings on these attacks it is very difficult to say. It may be that some dietetic error, such as an excess of fat or a diminution

of carbohydrate in the food, so that constipation or slight vomiting determine the onset of the acid intoxication. With the onset of vomiting the acid intoxication is increased. Chronic appendicitis with recurring subacute attacks must be very carefully excluded before a diagnosis of cyclical vomiting is made. Nevertheless, it is certainly not true to say (as has been stated) that all these cases are in reality due to disease of the appendix.

Treatment is on the same lines as those laid down for post-anæsthetic acetonæmia. Although they are rare, certain instances occur in which nothing seems able to check the vomiting, nor any amount of alkali to turn the urine alkaline, and death takes place.

1. **Salicylic Acid Poisoning.**—The toxic symptoms of the salicylate group of drugs are due to acid intoxication. Many symptoms which have been held to be the ill-effects of the drug, such as anemia, cardiac depression, mental depression, and albuminuria, are in reality due to the acidosis for which the drug is given. Headache and tinnitus are very rarely induced by salicylate in children. The real symptoms of salicylate poisoning are those of acid intoxication: namely, vomiting, drowsiness, anorexia, restlessness and delirium, acetone in the breath and urine, and in the worst cases fatal coma.

The amount of the drug given is of much less importance in the production of toxic symptoms than is the method by which it is given. It is easy to poison a child when giving 20 grains of sodium salicylate daily, and easy to avoid poisoning although 200 or 300 grains are being given each day. A fatal case has been reported on 20 grs. per day given over a long period; but this was before the true dangers of salicylic acid were appreciated. The toxic symptoms may be produced if the bowels are allowed to be confined, and if alkali in sufficient quantity to keep the urine alkaline be not administered with the salicylate.

Treatment.—An equal or a double dose of sodium bicarbonate may be given with the salicylate; and should they be proved necessary by the examination of the reaction of the urine, extra doses of alkali may be administered from time to time. Constipation must be avoided by the daily use of aperients, and in particular no increase in the dose of salicylate should be made if the bowels have not been well opened. Vomiting is not necessarily a sign that the child has an idiosyncrasy against salicylate, but should be treated by the temporary omission of the drug, which can be administered again in smaller doses after a lapse of twelve to twenty-four hours. It can then, as a rule, easily be given in increasing amounts.

Agonia, which cannot be given in combination with alkalies, is now liable for this reason to produce acid intoxication.

Should symptoms of acid intoxication arise, the salicylate should be immediately withheld, a purge given, and large doses of sodium bicarbonate or sodium citrate administered. The salicylate may be resumed if necessary, in a few days' time.

TESTS FOR ACETONURIA.

1. To some urine in a test-tube add a small quantity of freshly prepared sodium nitroprusside solution (5 per cent), and on to the top of the liquid pour some liq. ammonia, fort. Where this meets the urine, a mauve (maroon) ring will develop on standing, quite different in colour from the reddish-brown ring which is seen in an acetone-free urine.

2. *Legal's Test*.—Nitroprusside of soda is added to some urine as before. To this is added some liq. potash, and the contents of the test-tube become a crimson colour (due to creatinin). Acetic acid is now added in considerable quantity: with acetone present the fluid becomes darker in colour; without acetone, paler or colourless.

3. *Lieber's Test*.—To the urine is added some caustic potash, and then a few drops of an aqueous solution of iodine in potassium iodide (100:1) must not be used. A deposit of the yellow crystals of iodoform occurs, and the characteristic odour of that substance can be recognised.

SECTION IV.

INFECTIVE DISEASES.

1.—THE PNEUMOCOCCAL INFECTION.

Introductory.—The tendency towards generalization which is shown by all bacterial infections in children is perhaps of more importance in connection with the pneumococcal infection than any other. From this liability there arise many difficulties in diagnosis and prognosis which in adult patients are of considerable rarity.

Cases of generalized pneumococcal infection are seen in two clinical groups. In one the infection is generalized from the start. It runs a rapidly fatal course, and post mortem early involvement of the pleura, pericardium and meninges is found to be present in addition to the pneumonic consolidation in the lungs. Such cases are probably hopeless from the first; but their recognition is usually a matter of extreme difficulty, as death occurs before the development of signs other than those of pneumonia with a very intense toxæmia. In the second group, generalization occurs from some local source of infection, usually an empyema; and it is chiefly for this reason that the early detection and immediate evacuation of any such collection of pus are of such importance.

Difficulties arising in connection with this liability to generalization are seen almost daily in a children's ward. The temperature in a case of pneumonia has perhaps begun to fall or has reached normal, and then begins to rise again, and the child appears ill and poisoned. We have a variety of possibilities to consider: have we merely to deal with a pseudo-crisis, or has a fresh patch of pneumonia, an empyema, a paralytic pericardium, or an involvement of the ears, meninges or peritoneum arisen? Perhaps, with the renewed fever the child has become rigid, and various "meningeal" signs have developed. Here we may be dealing with a fresh attack of pneumonia with new or renewed cerebral symptoms, or with stitis media, pneumococcal meningitis, or with a slight overdose of strychnine. On the other hand, the meningeal signs may be due to tuberculous meningitis, and the pulmonary infection be in reality a tuberculous process or a pneumococcal infection occurring in a tuberculous lung.

With such difficulties as these, mistakes in diagnosis are almost inevitable; but to one source of error I would like to draw attention

at the outset of the consideration of the pneumococcal infection in children. The danger of regarding what is really a tuberculous process as pneumococcal, has been so much emphasized that it is now necessary to give a word of warning in the opposite direction. It is, I think often not sufficiently recognized how protracted and how erratic may be the course of a pneumococcal infection in a child, and without doubt more mistakes are made by regarding a process as tuberculous than by forgetting the possibility of tuberculosis. Such a mistake is full of danger, for the pneumococcal case, if diagnosed, may be rapidly curable. The signs and symptoms, by instance, of an undiscovered empyema may simulate closely those of pulmonary tuberculosis, but how greatly does the prognosis depend, in such a case, upon the accurate diagnosis of which infection is present.

The general constitutional symptoms of a pneumococcal toxæmia, if acute and severe, are generally very characteristic. They are here described under pneumonia; but it is to be remembered that this is the commonest, but not the only, cause of them. The clinical picture, so often said to be typical of pneumonia, is only so because pneumonia is the most frequent cause of a severe and acute pneumococcal toxæmia. Exactly the same symptoms may be seen in an empyema in which drainage has become temporarily blocked, or from time to time in an infected empyema, pericardium, or arthritis. Such symptoms may give a valuable clue to the type of infection existing.

PNEUMOCOCCAL PNEUMONIA.

In children practically all primary pneumonias are pneumococcal. A small proportion of consecutive or post-bronchitic pneumonias are caused by the same organism, but as they differ so much from the primary cases they are described separately (Section VI).

Etiology.—By a primary pneumonia is meant one which does not arise by extension from the upper respiratory tract. The disease starts suddenly; and throughout its course is accompanied by little or no generalized bronchitis. The infection of the lung is in most cases, if not in all, through the blood-stream.

Primary pneumonias in children are of two types, lobar and lobular. Both classes are pneumococcal in the vast majority of cases, and both run an exactly similar course.

Into the controversy which has raged round the question of the proportionate frequency of lobar and lobular pneumonias in infants, it is not really necessary to enter. Much of the difference of opinion has arisen out of the insufficient recognition of the primary type of bronchio-pneumonia. In this form bronchio-pneumonia begins suddenly and often terminates by crisis, simulating exactly that is to say, a lobar pneumonia. Nor do the physical signs in the lungs give an accurate method of differential diagnosis for a mixed

broncho-pneumonia may be distributed in a lobar form. Certainly it is that post mortem it is very rare to find a true lobar pneumonia, even in those cases which during life have simulated such a condition, and from this it is probably a correct deduction that the common primary pneumonia of infancy is broncho-pneumonic in type.

The controversy, we see, therefore, has been fused an unimportant matter. For it is of little importance to attempt to differentiate between a lobar pneumonia and a primary broncho-pneumonia; while it is of great importance to separate the primary from the consecutive, secondary or post-bronchitic, pneumonias.

Age-incidence.—This is shown in the accompanying diagram, based upon 127 primary cases in the wards of the Paddington Green Children's



FIG. 23.—INCIDENCE OF PRIMARY PNEUMONIA IN CHILDREN (127 CASES).

Hospital, London. It will be seen that the maximum incidence is in the second year of life, after which, speaking roughly, there is a rapid and continuous decline during the remaining years of childhood.

The Predisposing factors, when such can be traced, are similar to those seen in adults, namely, cold weather, exposure, chill, and injury, especially that of the head. One attack predisposes to others in the later years of childhood.

Symptomatology.—Primary pneumonia sets in suddenly, usually with vomiting, often with a convulsion, occasionally, in older children, with a rigor. The temperature rises rapidly to 102° or higher. There

are rapidity of breathing (tachypnea), dry cough, possibly some pleuritic pain, sore throat, headache, or pains in the back or neck. In some cases, particularly where an apex of a lung is involved, the child is unconscious and rigid from the start.

It is of great importance to be able to recognize a case of pneumonia without relying upon the presence of any physical signs in the lungs, for often these are late in their development.

General Symptoms.—As a rule, the aspect of the child is very characteristic. The face is flushed, and the skin burning and dry. Various cerebral symptoms may be present which are detailed below. Most suggestive are the changes in the child's breathing. The respiration-rate is quickened, often up to 40 or 50 per minute in an infant, and up to 30 per minute in older children. This, typically, causes comparatively little distress, and is better termed tachypnea than dyspnea. The respiratory movements are mainly abdominal. The respiratory rhythm is reversed, the pause occurring when the lungs are full instead of at the end of expiration. At the beginning of each expiration there is a short grunting sound. There is a frequent short dry cough, which is much increased when the patient is turned on to its side. The ala nasi may move but little, but it is to be noted that if the nostrils expand it is with each expiration and not inspiration. Herpes may be present about the nose or lips, occasionally appearing first on the inner surfaces of the latter. The tachycardia is very commonly present. pneumonia is the most frequent cause of this phenomenon in children.

In a few cases there are little tachypnea, no cough, and no pulmonary signs during the first two or three days of the disease. In the absence of cough, pneumonia can hardly be diagnosed with certainty. The cough usually develops by the third day, and in twenty-four hours more signs of pneumonia appear in the lungs.

As the disease progresses, the appearance of the patient may change. The child may become more pale, and slight cyanosis develop in the lips and cheeks. Signs of true dyspnea may become present. In infants in-curling of the lower lip during inspiration is an early sign of this. The respiration-rate becomes still further quickened. The cough becomes looser. During this stage we have the symptoms of severe toxemia and slight right heart dilatation.

Where the disease is running an unfavorable course, the symptoms of failure of the right ventricle become more marked. The pallor, cyanosis, and dyspnea are increased. In infants the mouth is opened and the head slightly withdrawn with each inspiration. The pulse becomes more rigid and feebler. The cough is very loose, and the breathing becomes noisy and rattling. At this stage death occurs.

The Cerebral symptoms of pneumonia are of great importance as they often tend to overshadow the clinical picture of the disease. In many cases there is considerable drowsiness present. This drowsiness is usually due to a profound toxemia, but sometimes to acid intoxication.

(p. 86), when it is associated with vomiting, restlessness, and an hunger. A violent and very active delirium is the most feature in some cases of pneumonia. This may mask the true nature of the disease; but in such a condition the possibility of pneumonia, especially of an apical pneumonia, should be uppermost in the observer's mind.

Still more puzzling may be the meningeal symptoms which may be present in pneumonia as the absence of inflammatory meningitis. These symptoms are more common in apical than basal cases, and to them the term *meningismus* has been applied. They probably depend upon a condition of meningeal oedema. The *nuxie cérébrale* has already been mentioned, and is frequently present without any of the other nervous signs. There may be generalised rigidity, the limbs being stiff, and Kernig's sign (p. 13) positive. The head may be retracted, although not to an extreme degree, yet as much as or more than in tuberculous meningitis. There may be tremulous movements in the limbs, as in a case of vertical meningitis. A definite squint may be present. The child may be quite unconscious, with the abdominal reflexes absent. The knee-jerks are usually brisk, but during the height of the toxæmia are often temporarily lost. In children under five or six years of age, extensor plantar responses may be obtained while they are in this condition. The breathing may show some tendency towards becoming grouped; but this does not go so far as to show definite periods of apnoea. Convulsions may be present at the invasion stage of pneumonia, but in the absence of meningitis do not continue.

The symptoms of meningismus therefore closely simulate those of meningitis. Bulging of the anterior fontanelle, optic neuritis and well-marked grouping of the breathing, are not seen in meningismus; while the cerebrospinal fluid, although increased in amount and under pressure, is clear and contains no excess of cells, and is practically always free from organisms.

The Abdominal symptoms of pneumonia are worth more consideration than is often given them by students. Vomiting at the start of the disease is very common; in scarlatina this initial vomiting is very constant, in pneumonia it is very frequent. It may continue during the first week of the disease, in which case it is usually associated with acetonaemia. The severity of the cough may account for an occasional vomit at any period of pneumonia. In some cases there is well-marked gastritis present, sometimes of pneumococcal origin, sometimes possibly due to such drugs as ammonium carbonate. Diarrhoea may be troublesome in some infantile cases, but this is uncommon in primary pneumonias; constipation is, as a rule, present. The spleen tends to become enlarged in any protracted or severe pneumonia; it is not necessarily, therefore, a sign of the disease being tuberculous.

At the onset of some cases, usually those in which the base of the right lung is involved, there may be pain and even apparent tenderness in the right iliac fossa. These symptoms, with the vomiting and

constipation, may give rise to a mistaken diagnosis of acute appendicitis.

The most dangerous abdominal symptom in pneumonia is that of general distention. This may develop in any severe case by the end of a week and when at all marked adds considerably to the dyspnea. Its appearance is always a danger signal. In this connection the possibility of pneumococcal peritonitis may require consideration.

The Temperature of pneumococcal pneumonia is usually sustained at first, then begins to break slightly before it makes a definite descent to the normal. Less regular temperatures, sometimes remittent in character, are seen in younger children. The fall may be by crisis or by lysis. A pseudo-crisis is distinguished from a true crisis by the fact that there is no diminution of the respiration-rate with the fall of the temperature. Within twenty-four hours of a pseudo-crisis, the temperature falls by crisis or begins to fall by lysis.

With the descent of the temperature to the normal, the patient quickly regains a look of comparative health; but it is not uncommon for the child, after a severe attack, to remain very toxic in appearance, and for the tongue to remain dry for twenty-four or forty-eight hours after a crisis. Such conditions are not necessarily due to a developing mycæmia.

The Heart shows much the same changes in children as in adults. Dilatation of the left ventricle is very unusual in pneumonia, and generally indicates that the disease was ushered in by influenza. The right side of the heart is very frequently dilated, and where this occurs there are increased dyspnea, cyanosis, moist rales at the bases of the lungs, and enlargement of the liver. The extent of the deep cardiac dullness to the right must therefore be carefully and regularly estimated by light percussion throughout the disease.

The daily observation of the deep cardiac dullness is also particularly necessary in children for another reason. In them, especially in those under the age of five years, a persistent pericarditis is by no means uncommon; and in a condition so notoriously difficult to diagnose as this, a series of observations of the changes in the area of deep cardiac dullness may be of considerable value.

It must be noted, that in a few cases there is a slight displacement of the heart away from the sternum inq. This, which is probably due to the balance of the diaphragm being upset, is not more than slight in amount, but can undoubtedly occur in children. Clubbing of the fingers is rarely present in an uncomplicated pneumonia.

The Urine shows the same changes as in adults. In addition, acetonauria is not uncommon. Bacteriuria is rare.

The Blood changes do not differ from those seen in pneumonia in older subjects.

Pulmonary Signs.—As is well known, no definite signs of consolidation may appear in the lungs until the disease has been present for several

days. As a rule, the pulmonary signs are present at least two or three days before the crisis; but they may even be deferred until after the temperature has fallen to normal although this is very rare. Cases with delayed physical signs seem to run rather in groups—when one is met with others are likely to be seen. The development of a cough usually precedes the appearance of the pulmonary signs by a day or two.

Most commonly, however, the signs in the lungs appear within twenty-four hours of the onset of the infection. The earliest signs are slight impairment of the percussion note associated with diminution of resonance. Over this area, within twelve to twenty-four hours the signs change to the more definite ones of dullness, bronchophony, and bronchial breathing. Crepitations are heard at the end of inspiration when the child cries in most cases; but it is not very uncommon for a pneumonia to run its course without any moist sounds being detected in the consolidated pulmonary area. The vocal resonance occasionally shows some argophonic timbre.

The case with which in children pleural oscillation signs may be conducted and thus become audible over healthy lung-tissue, must not be forgotten.

In a primary pneumonia there is little or no generalized bronchitis. With dilatation of the right side of the heart, rales appear at the bases of the lungs.

The expectoration is so commonly swallowed in the case of children that its characters are of very little importance.

In a few cases, for a day or two before and after the crisis, the dullness over the pneumonic area may be so absolute and so resistant in character as strongly to suggest the presence of pleural effusion.

The right lowest lobe is the most common seat of pneumonia. Apical and basal cases occur in the proportion of two to five (Goodhart).

As a rule, the pneumonic process can be localized in the lungs without difficulty; but owing to the frequency of cases with delayed development of the pulmonary signs, it is very necessary to be able to recognize pneumonia from the general appearance and symptoms of the child. Often it is easier to make a diagnosis of pneumonia than to state which part of the lungs is involved. Where the cerebral symptoms are marked, the apices of the lungs should be examined with special care.

Course.—The infection most commonly comes to an end within six to nine days, the crisis occurring most frequently on the seventh day. Abortive cases lasting ten, two or three days occur, but they are hard to recognize with certainty. Probably in some of them the condition is one of influenza pneumonia or of collapse of lung.

On the other hand, protracted cases are far from uncommon. In a series of 127 cases of primary pneumonia which I collected from the records of the Paddington Green Children's Hospital, no less than 20 per cent showed that the temperature had not begun to drop by

the end of the tenth day. Such a proportion must be regarded as exceptionally high, and is probably explained by the greater tendency for a protracted case to seek admission to hospital.

The prolongation of the fever may be associated with (1) *Protracted non-spreading pneumonia*; (2) *Protracted spreading or recurrent pneumonia*; (3) *Imperfect resolution*; (4) *Pneumococcal manifestation other than pneumonia*. Of these the first two groups include over two-thirds of the cases.

1. **Protracted Non-spreading Pneumonia.**—These do not differ from ordinary cases except that their course is prolonged. The pyrexia may go on for fourteen days unaltered; but in most, either recovery

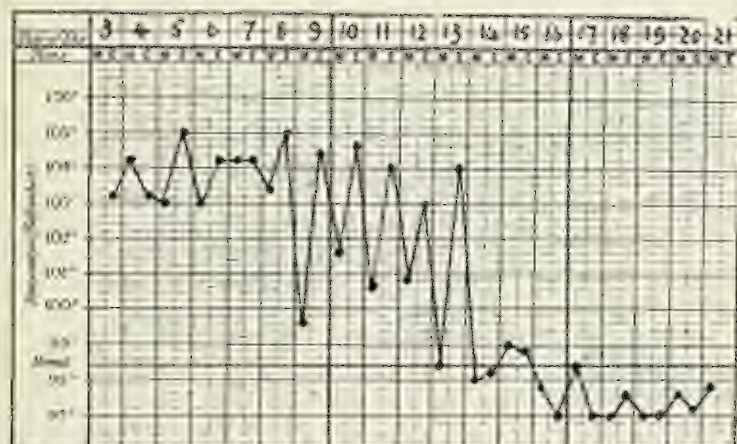


FIG. 24.—CHART OF PROTRACTED PRIMARY PNEUMONIA IN A CHILD (AGE 1½ YEARS).
CHART PREPARED BY PHOTODUPLICATION.

or death occurs by the end of the twelfth day (*Fig. 24*). The death-rate in this group is about four times as great as in cases of unprotracted primary pneumonia.

2. **Protracted Spreading or Recurrent Pneumonia.**—This group is the largest of the four. The pneumonic consolidation passes from one area of the lung to another. The second attack is usually of shorter duration than the first, and may occur while the temperature is at its height, or while it is falling. It may also take place after the temperature has been normal for several days, but such cases hardly come into the consideration of protracted pyrexia in pneumonia. As a rule there is only one secondary focus of pneumonia, but two or three may develop. With the second attack, several symptoms may reappear or may now develop for the first time. In some cases the physical signs of the second attack are late in appearing,

The mortality in this group is about three times as great as in the uncontrolled cases.

Mention must here be made of certain relapsing cases in which areas of consolidation develop in various parts of the lungs over a period of many weeks. With each fresh focus there is a rise of temperature, while the paroxysmal periods are separated from each other by three or four days in which the temperature is normal. It is probable that such cases as these are associated with an influenza infection.

3. **Prolonged Fever associated with Imperfect Resolution.**—This is not very common in primary pneumonias. The signs in the lungs do not clear up, and the fever persists. It may become swinging in type, and in some cases is probably associated with a deposit of lymph on the surface of the pleura.

4. **Prolonged Fever associated with Pneumococcal Manifestations other than Pneumonia.**—Although we have here all the conditions such as empyema, pyopneumothorax, otitis media, meningitis, peritonitis, and others, yet such do not often cause the fever of a pneumonia to be prolonged beyond ten days. The reason is, that these bacterial infections usually either arise early and cause the death of the patient within a short time of the onset of the disease, or they develop after the temperature has been lowered for a few days. In a few cases only, the initial temperature of the pneumonia is kept up for over ten days by the occurrence of one of these pneumococcal manifestations.

Complications and Sequelæ.—Apart from the various pneumococcal metastases, which are much more common in children than in adults, there are very few conditions to be mentioned here. Jaundice occasionally develops in the course of a pneumonia, but is more commonly associated with empyema or purulent pericarditis. Bradycardia, cardiac weakness, and anemia are seldom of serious import. Fibrosis of the lung is the gravest sequelæ. It is, however, very uncommon. Delayed resolution is not so rare, for all such cases do not necessarily go on to fibrosis; resolution, although delayed, may ultimately be complete.

Diagnosis.—The diagnosis of pneumococcal pneumonia is often by no means easy. The chief difficulties arise in connection with delayed pulmonary signs and marked cerebral symptoms in the early part of the disease, and from irregularities in its course and atypical signs in the chest during the latter stages. From a consecutive pneumonia (q.v.) the primary disease is, as a rule, easily distinguished by its onset, physical signs, and course.

We are always to be prepared to diagnose pneumonia in the absence of any pulmonary signs. The general symptoms have been fully detailed (p. 89), and are usually quite distinctive.

From Scarlatina.—During the first twenty-four hours of illness,

pneumococcal pneumonia is closely simulated by scarlet fever. In both infections an initial vomit is very common; its absence is in favour of pneumonia. In both there is the sudden onset of high fever and the dry burning skin. The chief differences, in the absence of pulmonary signs, are concerned with the pulse and respiration rates. In pneumonia it is the respiration rate which is much increased, while in scarlatina the quickening of the pulse is the more marked. Movements of the air mast, grunting expirations, reversed respiratory rhythm, and cough, are all very strongly in favour of pneumonia. These, however, may be absent. Any severe follicular infection points towards scarlatina. Where the diagnosis cannot be made, and as a rule the details mentioned are sufficient to give clear indication, waiting for another day will generally settle the matter. Transient erythematia may occur in pneumonia, but the true rash of scarlatina is quite different from these.

From Acute Meningitis.—Where the cerebral symptoms are marked, pneumonia may simulate the various forms of acute meningitis. These symptoms have been described on page 29, and both the positive and negative signs of meningitis have been there discussed. It is necessary here to add only a few further remarks. Tuberculous meningitis does not usually simulate pneumonia very closely during infancy, and may be generally differentiated by its onset, the bulging of the fontanelle, and deep drowsiness. In older children, however, the earliest stage of this form of meningitis, with its admixture of drowsiness and violent delirium, may easily be mistaken for pneumonia. Posterior basic meningitis is as a rule distinguishable from pneumonia by the bulging of the anterior fontanelle, and the blindness and staring of the eyes. It must not be forgotten, however, that pneumonia, usually of a post-bronchitic type, may usher in a meningococcal meningitis. Pneumococcal meningitis is generally a complication of pneumonia, and it is often extremely difficult to exclude its presence.

The signs which point to any form of meningitis are convulsions repeated during the course of the disease, bulging of the fontanelle, optic neuritis, marked grouping of the breathing, but of more importance than any is the abnormal condition of the cerebrospinal fluid. Lumbar puncture should be undertaken in any case of doubt. Where meningismus only is present, it usually affords some relief, while it renders the diagnosis certain.

Where there is a history of recent injury to the head, the differential diagnosis between pneumonia and meningitis is often made increasingly difficult.

To sum up, we may say that it is rarely wise to make a diagnosis of meningitis where, by the general symptoms or by the physical signs in the chest, we can conclude that there is active pneumonia present. Even should pneumococcal meningitis be present, it usually becomes more clearly recognizable before death occurs. It is in connection with meningitis that it becomes so important to be

able to recognize pneumonia in the absence of any signs of consolidation in the lungs.

From Otitis Media.—Since otitis media is usually due to the pneumococcus, the general symptoms will be much the same as those of pneumonia, while of course the two conditions frequently co-exist. Signs of pain in the ear, such as head-rolling, head-banging, or the child putting its hand to the ear, point to otitis media. No local signs need be present in the ear at first.

From other causes of Delirium.—Very violent delirium in children is seen in pneumonia, typhoid fever, cholera and atropine poisoning. That due to pneumonia is the commonest, and is usually easily recognizable by the changes in the respiratory rate and rhythm. Consolidation at the apices of the lungs is likely to be present or to develop.

From Acute Appendicitis.—As in adults the onset of pneumonia may closely simulate that of typhoidism. Increase of the respiration-rate out of proportion to that of the pulse-rate, and the presence of cough, are in favour of pneumonia. The focal signs in the right iliac fossa are rarely very marked in pneumonia, and much rigidity, hyperaesthesia and immobility here point towards appendicitis. The chest, particularly the base of the right lung, should be very carefully examined.

From Cyclical Vomiting.—Occasionally in pneumonia the symptoms of acid intoxication (p. 81) are very marked, and the breath and urine contain much acetone. With ordinary care, however, there is no difficulty in recognising the pneumonia underlying the condition by the great increase in respiration-rate which may thus co-exist with the panting breathing of asphyxia. The cough and other signs also make the diagnosis clear. In cases of doubt the administration of alkalis is almost sufficient to render the urine alkaline, will cause the cessation of such symptoms as are due to acid intoxication.

From Tuberculosis.—Pneumococcal pneumonia and intrathoracic tuberculosis frequently simulate each other very closely. Speaking generally, during the early part of the infection, tuberculosis may be mistaken for pneumonia, while during the later stages, a pneumococcal infection may be mistaken for tuberculosis.

At the beginning of an illness, what is in reality an acute tuberculous infection of the lung may be mistaken for a pneumococcal condition. The dyspnoea and sustained fever are common to both, but the tuberculous cases can usually be distinguished by the pulse and prostration of the child, while the polymuclear leucocytosis is absent. In other cases a tuberculous pleural effusion, of which the onset may be exceedingly acute, may at first simulate pneumonia. With an increasing collection of fluid, however, the diagnosis becomes clear.

Later, it is usually the protracted nature of the disease that raises the suspicion of tuberculosis. I have already ventured to warn the reader against diagnosing tubercle too readily in such a condition,

and I have given some account of the various causes of the prolongation of the fever in pneumonia. Every effort should be made to exclude the possibility of an unfocalized empyema. One exploration with a needle in a doubtful case is not sufficient for this purpose. The white cells in the blood should be examined; not only their total number, but their proportionate numbers should be taken into account. In a pneumococcal condition they are increased in number, and the proportion of polymuclear cells is high. In tubercle they are also increased to some extent, but the lymphocytes show the largest increment. A good skiagraph may be of considerable service in helping to differentiate between empyema and pulmonary tuberculosis. One further point is of some value. In infants a prolonged fever with pulmonary signs is not likely to be tuberculous in origin unless symptoms of heringeal tuberculosis supervene. It is rare in them to find a very active tuberculosis remaining for long localized to the lungs.

Where the pulmonary signs remain confined to the left lung, the possibility of a pericardium with compression of the lower lobe of the left lung, will have to be very carefully considered.

It is to be remembered that, with an undrained pneumococcal abscess the patient may come to have a very tuberculous appearance, wasted, pale, with a waxy although flushed complexion, and with a growth of downy hair on the temples, lips, and back. Such, then, is not sufficient to substantiate a verdict of tuberculosis in such a case.

From Acute Pyelitis.—This condition, which is not uncommon in female infants, is usually mistaken at first for pneumonia owing to its sudden onset with high fever, tachycardia, and generalized rigidity. The occurrence of remissions in the temperature, rigors and faint-ness is in favour of acute pyelitis. The urine should be carefully examined for *B. coli communis*. Later pyuria and symptoms of cystitis develop.

From Empyema.—The diagnostic points in connection with pneumonic consolidation and empyema are considered under the latter condition (p. 184).

Prognosis.—The death-rate in primary pneumonia in children is considerably less than in adults. In the series of 127 cases I collected from the Paddington Green Children's Hospital, I found that death occurred in 24 cases, or 19.0 per cent. This figure, which covers all cases, is probably high, as those which are admitted are of the worst type, and often occur in badly nourished children. Dr. Riviere gives the percentage of mortality as 6.6.

The death-rate varies very considerably, according to the age of the child and the length of the attack.

Primary pneumonia in children over the age of two years is itself rarely fatal. In the 83 cases occurring between the ages of two and twelve, only 1 (1.2 per cent) was fatal. In infancy, however, the mortality in 44 cases amounted to 14, or 31.5 per cent.

When the fever lasts for longer than two days, the danger of the disease is much increased. In 35 protracted cases of all ages, 10 died (30 per cent); while in the 45 unprotracted cases only 4 (9 per cent) ended fatally.

Death during infancy usually arises either from the profound toxæmia or from a generalized pneumococcal infection (septicæmia), of which the pneumonia is only a part. In older children the chief danger arises in connection with some pneumococcal infection, such as an erysipelas, from which a generalized infection or fatal metastases originate. In a few cases, however, in these children, as in infants, the infection is generalized from the start.

Treatment.—The child should be put to bed in a warm, well-ventilated and large room, the windows of which are to be kept open day and night, except when the patient is uncovered for any purpose. The clothing should be light, loose, and warm, and if the temperature is high, the bedclothes may be raised on a cradle and the patient covered by a single blanket. Care must be taken not to let the child lie with its lower extremities uncovered. In cold weather, a jacket of Gossier tissue may be ordered. The use of bed curtains is not to be recommended, as plenty of fresh air is a necessity to the child. For the same reason, the room should be kept as empty of people as possible. The mouth must be kept moist and clean by means of antiseptic washes.

The diet should consist of milk, to which beef-tea, eggs, and the lighter farinaceous foods may be added where required. It should be given in as large quantities as can be digested. As much water as is asked for may be allowed except in cases where there are symptoms of right heart-failure, in which it is wise to limit the amount of fluid given.

At the outset of the treatment, the bowels should be opened by a moderate dose of calomel (1 grain), followed by a saline aperient. Throughout the disease constipation must be avoided, as it favours abdominal distention. Small doses of calomel with soda, and the salines are usefully continued during the infection.

Cough mixtures should not be given as a routine, for they help to upset the digestion. They are rarely of use during the early stages of pneumonia. Later, where stimulating expectorants are required, ammonium carbonate is of most value. With it, speccanumia wine may be given. Some Dover's powder, or the compound tincture of camphor, may be given during the earliest stages if the cough is very troublesome or if pleuritic pain is present.

When cough mixtures are not indicated, a fever mixture containing liquor ammonii acetatis and potassium citrate or spiritus ætheris nitrosi may be given. By keeping the skin moist, it confers to the patient's comfort.

The temperature may be reduced by tepid sponging. As a rule

this is undertaken when the temperature reaches 104° ; but sponging may be ordered at a lower temperature should the child become restless and uncomfortable. Ice-bags covered in cotton-wool may be suspended within the bed cradle, in order to surround the child with cool air. Of more avail is the application of an ice-bag to the pleuritic area of the lung. This not only tends to reduce the temperature and to quiet delirium, but relieves some of the congestion in the affected area of the lung. It should be applied in the way indicated on page 377, and with the same precautions. Hot water bottles should be put to the patient's feet.

Pleuritic pain may be treated by an ice-bag, leeches, poultices, or by strapping the side of the chest. In the absence of signs of right heart-failure, there is no objection to the use of opiates in small doses; they are best administered in the form of Dover's powder or tinct. camph. co. In infants it is wisest to avoid their use altogether.

Insomnia and restlessness are usually due to pleuritic pain, high fever, or right heart-distress, and it is rarely necessary to treat them by the use of direct sedative drugs. In a few cases they are due to an associated acetonaemia, and should be treated by alkalis. A drachm or two of brandy at night is perhaps the best specific. Chloral and bromides may be used in cases of severe delirium. Lumbar puncture may be of benefit in relieving the symptoms of meningismus.

The treatment of the heart is of prime importance. Right heart-failure is shown by the extension of the deep cardiac dullness to the right, by the increase of dyspnoea, the onset of cyanosis, oedema of the lungs, and enlargement of the liver. Such signs as these directly indicate the use of leeches. The improvement which follows the application of leeches is often extremely definite, and they may be the means of saving life. I am sure that no one who has made trial of them in pneumonia would willingly dispense with their use. They are best applied over the right costal margin. One or two may be ordered for an infant, three or four for an older child. Should they refuse to bite, a drop of blood from one's own finger should be smeared on the patient's skin. The bleeding from the bites may be encouraged if necessary, by the use ofomentations, and may be checked by the application of an ice-bag or by the pressure of a band. It is rarely wise, however, to stop such bleeding, as it is usually beneficial. Pallor is not necessarily a contraindication to the use of leeches.

Combined with the relief of the right heart by leeches, we may make use of cardiac stimulants. Of these the best is strychnine. For a baby under a year, a minute of a quarter-strength solution of the hydrochloride of strychnine (i.e. 1-2000, given hypodermically every four hours, is the full dose, and cannot be kept up as a rule for very long. Alternating with the strychnine, a mixture of digitalin and atropine may be given hypodermically. Brandy is of great service, and may be given freely in the feeds, up to an ounce in the twenty-four hours for a child of one year.

Oxygen is seldom of more than temporary benefit. It may be administered for five minutes every half-hour, but should it cause fright, as it often does when given within six inches of the child's face, the gas may be allowed to escape continuously from the cylinder at the foot of the bed. When delivered close to the mouth the gas should be warmed by being bubbled through hot water. If passed through absolute alcohol its stimulating effect is increased.

The condition of the abdomen must not be neglected. Digestion calls for the use of aperients, such as repeated small doses of calomel, and for more digestible food. The milk may be further diluted, diluted, or peptonized.

The most dangerous time in pneumonia is during the day or two preceding the fall of temperature. The crisis itself is rarely fatal in children.

In the treatment of pneumonia in young subjects, three rules should be borne in mind. First, never withhold stimulants for fear of worse symptoms arising; children so quickly pass out of hand, that all measures should be used to the fullest extent that is indicated at the moment. Second, be perpetually on the look out for the various pneumococcal complications, such as empyema, purulent pericarditis, and the others which are so much more common in children than in adults. Third, never lose hope; careful attention from hour to hour will often be rewarded by the successful issue of a seemingly hopeless case.

Convalescence. This is usually rapid in the absence of any complications. Fresh air, good food, and tonic drugs such as cod-liver oil, iron, and arsenic, are the chief measures in promoting a complete restoration to health.

PNEUMOCOCCAL PLEURISY.

Dry Pleurisy.—Pleuripneumonia occurs in children, as it does in adults. During the first few days of pneumonia there may be considerable pleuritic pain, accompanied by a rough friction audible on auscultation. Occasionally, pleurisy complicates bronchitis.

The treatment of this form of pleurisy has been already detailed (p. 99).

Pleurisy with Effusion.—A clear effusion containing a few pneumococci is seen in connection with pneumonia on very rare occasions. Where it occurs there is a great probability of the effusion becoming purulent, but this is not an invariable result. The case should be watched for a few days, and if the signs of fluid do not clear up, a needle should again be inserted, to ascertain the nature of the effusion. If still clear, it may be aspirated or left, but if now purulent it should be evacuated in the usual way. These cases are, however, so uncommon that for all practical purposes a pneumococcal pleural effusion means an empyema.

Empyema.—The vast majority of empyemata in children are due to the pneumococcus. Dr. Kiriakow found this organism alone in 85.7 per cent of the cases, while associated with streptococci and staphylococci, it was present in altogether over 92 per cent. The non-pneumococcal cases are rather less readily cured than are the pneumococcal, but otherwise the two groups of cases are similar.

EMPHYEMA.

Symptomatology.—An empyema may begin to develop with the onset of the pneumonia; in which case, should the child survive, the symptoms fail to disappear at the period of resolution of the lung condition. On the other hand, an empyema may arise as the temperature from the pneumonia is beginning to fall, or after it has been normal for a few days. In some cases the symptoms of an empyema are very latent, and the child may come under observation for wasting.

Occurring as is usual in obvious association with pneumonia, the symptoms of empyema are raised temperature, often hectic in type, increased pulse and respiration-rates, cough, pallor, wasting, and a high leucocytosis. The cough may be paroxysmal in type. Clubbing of the fingers arises rapidly, and as the collection of fluid becomes larger, the dyspnea increases and cyanosis appears. Attacks of transient high fever with much dyspnea and sweating, may occur from time to time if a large dose of poison be absorbed suddenly.

In other cases, where the empyema is small and does not increase in size, the symptoms are much less suggestive. With some irregular fever, pallor, and perhaps slight clubbing of the fingers, wasting is the most prominent symptom. Such cases may be mistaken in infants for simple marasmus, and in older children for pulmonary tuberculosis.

If not drained, an empyema may be expectorated, or may after many weeks point, the swelling usually appearing in the front of the chest under the edge of the pectoral muscles. Some of the more latent cases may probably become abscessed or calcified, but we know very little of such occurrences in children. More often, a neglected case



FIG. 15.—EMPHYEMA PLEURIS IN CENTRAL NERVOUS.

makes its way into a bronchus and is expectorated, sometimes with immediately fatal results.

Physical Signs.—Of the signs in the chest, the most important is the resistant character of the dullness over the affected area. The upper border of the dullness is a sinuous line, high in the axilla. The resonance over the dull area may be absent, often it is only diminished towards the base of the lung. In some cases the breath-sounds are loud. The relative diminution of the breath-sounds at the base of the lung is therefore of more importance than their actual loudness or softness. Where emphyse, the breath-sounds are in the great majority of cases bronchial in character, being conducted by compressed lung. The vocal resonance and fremitus tend to be diminished or absent. Stothair resonance, egophony, Froese's sign, displacement of viscera, and bulging of the intercostal spaces may be present, as in adults. The significance of the various signs is discussed under the diagnosis of empyema. Clubbing of the fingers has already been mentioned. Effusion of the chest-wall is a rare sign.

When a large effusion is present, there may be signs of oedema of the opposite lung.

When an empyema is suspected but cannot be localized, it may be useful to recall the situations in which such a collection of pus is most difficult to find: these are—between the lobes of the lung, at the apex, between the lung and the heart, and between the lung and the diaphragm. It must, however, be remembered that the supposed collection of pus may be in the pericardium and not in the pleural cavity.

Diagnosis.—This may be most conveniently discussed under two headings: (i) *The diagnosis of the presence of fluid; and* (ii) *The diagnosis of the nature of the fluid.*

1. *The Presence of Fluid* may be easily recognized where there is a large amount collected in the pleural cavity; the resistant dullness, the diminished breathing becoming absent or diminished at the base, the alterations in vocal resonance and fremitus, bulging of the intercostal spaces, well-marked egophony and Stothair resonance, with much displacement of the viscera, dyspnoea, and clubbing of the fingers, all these tend to make the diagnosis very clear.

In other cases the difficulty of diagnosis is extreme, and frequently it is not possible to go further than to come to a decision as to the propriety of making an exploratory puncture of the chest.

Of the signs in the chest, the most important is the resistant character of the dullness. In the absence of this, fluid is very unlikely to be present, while it may be the only definite sign pointing to pleural effusion. It may be simulated very closely for the dullness of a pneumonia lung at or shortly after a crisis, by a lung solid from the rupture of a caseous bronchial gland into a bronchus, and occasionally by a pneumothorax.

It is well known that in children the breath-sounds may be loudly heard through fluid. While this is so it is important to note two points in regard to the breath-sounds. Firstly, where vesicular they are bronchial in type: there are very few exceptions to this rule. Secondly, that any definite diminution in the amount of air-entry as the base of the lung is approached is very suggestive of pleural effusion. It is quite possible for the breath-sounds to be audible right down to the base, but that they are nevertheless of less intensity at the lower part of the dull area than above, is the point of importance. To absence of air-entry at the extreme base, little need can be paid, as there is often here a fringe of collapsed lung about an inch deep in cases of pneumonia.

Esophagus, if well marked, is an important sign of fluid, but both this and Skratke resonance may be heard in their slighter forms in consolidated lung.

Signs of compression of the lung are usually present at the apex, and may be of some help; but they may be so marked as to suggest emphysema, and thus may cause a mistake in diagnosis.

Displacement of the heart away from the diseased side is a very valuable sign. It must, however, be allowed that slight displacement in this direction may occur with consolidated lung, as in pneumonia. If marked, the sign is one of the greatest importance, as it is only produced by liquid or air in the pleural cavity.

Freese's sign and bulging of the intercostal space, as a rule, only seen in large effusions which present little difficulty in diagnosis.

A skiagram in difficult cases may be of considerable help, particularly in interlobar empyemata. By this means also pulmonary tuberculosis may sometimes be excluded.

Puncture of the chest-wall by a needle, forms the last method of diagnosis. A frequent mistake is to insert the needle in too low a space, so that it traverses only the thin and poorly obliterated end of the pleural cavity. Another error is to make use of too thin a needle. Care should be taken to insertion suction as the needle is withdrawn, at once or two at first being inserted too far, and thus has passed through the fluid into the lung. In difficult cases it is wiser to give a general anæsthetic and make a thorough examination, puncturing the chest two or three times if necessary, so that if no fluid be found, an empyema may be excluded with fair certainty.

An interlobar empyema is very difficult to find. As a rule it comes to the surface of the lung posteriorly, along the line between the lobes. An area of dullness, rather oval in shape, may be found traversing the lobe of the lung obliquely. An area of modified resonance may sometimes be mapped out at the base of the lung next to the spine, instead of by the root of the lung, as is the case in ordinary empyemata. In the interlobar type, the breath-sounds may not be bronchial in character. Variability of the pulmonary signs, due to collapse of lung, is often a feature of these cases. A skiagram may be of considerable value.

†An apical empyema is fairly easily diagnosed if its possibility is borne in mind.

From Pneumonia.—In this connection the chief difficulty lies in excluding the presence of an empyema during the day before and a couple of days after the crisis in pneumonia. At this period of a pneumonia, in the absence of an empyema, the dull area of the lung may become very resistant to percussion, the temperature swinging in type, while the toxic appearance of the child may remain. It is well, therefore, so long as the patient's condition is not getting worse, to postpone the exploration of the chest for a day or two. Should the temperature become persistently raised, the leucocytosis of the blood increase, or the cough become paroxysmal in character, an empyema is probably developing.

From a Caceous Lung due to a Ruptured Bronchial Gland.—Practically every sign of pleural effusion may be given by this condition, and the differential diagnosis is one of great difficulty. It is, however, a condition which is very much less common than is an empyema, and is so very rare on the left side that the signs being on this side are much against such a possibility. The condition is rarely recognizable until repeated explorations have excluded the possibility of empyema.

From Thickened Pleura and Chronic Pulmonary Tuberculosis.—It is to be remembered that the symptoms of chronic pulmonary tuberculosis and an empyema may be exactly the same. The physical signs in this condition show that the dullness is not so resistant in character, and the breath-sounds, although they may be diminished, are seldom of the bronchial type. Other signs of pulmonary tuberculosis should be carefully looked for. Enlargement of the spleen may be present in both conditions; when great is absent, it is in favour of tuberculosis. In cases of doubt a needle should be freely used, for to leave an empyema may be a matter of the greatest danger.

From Pneumothorax.—Exceptionally, the pressure of the air within the pleural cavity is so great as to give rise to resistant dullness. As a rule, in such a case the diagnosis is only made when a needle is inserted into the chest. A pyo-pneumothorax is not uncommon.

From Pulmonary Abscess.—This is not as a rule possible until an operation is performed. The two conditions frequently coexist.

2. *The Character of the Fluid* is usually best settled by the withdrawal of some from the chest. When a pneumonia has been watched through its course, and is followed by the development of pleural effusion, the purulent nature of the fluid is fairly certain. A history of pneumonia is a less satisfactory guide. It must be remembered that a tuberculous pleural effusion may develop so rapidly and with such acute symptoms, as to simulate pneumonia.

The signs in the chest are the same in purulent and serous cases. Enlargement of the superficial intercostal glands (p. 125) is much more common in the serous than in the purulent cases, but its absence is a sign of no importance. An examination of the blood affords valuable

help: a polymuclear leucocytosis and a lymphocytosis being found in the purulent and serous effusions respectively.

In the absence of a blood-count, the diagnosis of the character of the fluid is notoriously uncertain; and as in many cases where everything points to one form of fluid, the other is found, it is better as a general rule to withdraw some of it by puncturing the chest.

Complications and Sequelæ.—The complications of empyema, which in reality constitute the chief dangers of the condition, are the other forms of pneumococcal infection, parietal pericarditis, meningitis, peritonitis, and pneumonia. Mediastinal suppuration and cellulitis, giving rise to an extra-pleural abscess, are rare complications.

The sequelæ of chief importance is that of a persistent sinus due to deficient expansion of the lung. Such must not be neglected, and will require further surgical treatment. Extirpation of senile portions of several ribs may be necessary. Less often, pulmonary fibrosis with bronchiectasis, thickened pleura and falling in of the chest-wall may result from a neglected empyema. Amyloid disease is a rare sequel, and may arise from either a neglected empyema or a persistent sinus.

Course and Prognosis.—An empyema, if undiscovered, usually leads to the death of the patient by the development of complications, by emaciation, with or without signs of amyloid disease. The empyema, however, may be spontaneously evacuated through the chest-wall or by rupture into a bronchus although, as a rule such an evacuation is incomplete. Rarely it may become dried up and calcified.

Death from an uncomplicated empyema is very rare. The liability to fatal complications is increased where the empyema arises with the onset of pneumonia, and where the patient is very young. In infants under the age of one year the condition is extremely fatal. For the same reason, the more promptly an empyema is recognized and drained the better is the prognosis. The length of time for which the empyema has been present is of more importance than its size. In large empyemata, sudden death during the operation is not very rare, a point alluded to later.

A double, even a triple empyema may exist without fatal complications ensuing, although naturally the liability to them is here much increased.

In pure pneumococcal cases the outlook is much better than where a mixed infection is present. A pure streptococcal infection is uncommon, but is by no means necessarily fatal. Such cases are, however, less favourable than where only the pneumococcus is present. A pure staphylococcal empyema is usually part of a pyæmia secondary to acute osteomyelitis, and for this reason the outlook here is extremely bad.

Treatment.—In order to diminish the liability to complications and to give the lung the best chance of complete expansion, immediate evacuation of the empyema should be practised.

As a general rule, it is best to resect a portion of a rib. Where the patient is very seriously ill, a simple incision may be preferable. When the case is seen for the first time with a very large collection of pus, much dyspnoea, and oedema of the opposite lung, it may be wise to separate the chest and allow the child to have a good night's rest, and to complete the operation on the following day.

During the operation the child should not be allowed to rest on the wound side. Not only does this embarrass the breathing, but should perforation into a bronchus occur during the operation, the healthy lung may be flooded with pus. The child should therefore be brought well to the edge of the table, and the surgeon should work as much as possible from the under surface.

It is usually wisest to insert an exploring needle into the chest before making an incision, even if the pus has been only recently localized. A portion of the eighth or ninth rib in the posterior axillary line is best chosen for resection, but this must depend upon the localization of the pus. An apical empyema may be opened from in front or from the apex of the axilla. A drainage tube of large calibre (5 mm. or of equivalent) should be inserted, and as a rule no irrigation should be done on the table.

As soon as drainage is established, the wound should be covered by the hand, which is quickly replaced by a thick layer of dressings. By these means, evacuation takes place gradually, and the danger of sudden collapse may be avoided. In a large empyema, the sudden escape of the pus from the chest, aided by coughing, is not seldom a source of great danger, and may be fatal.

The further surgical treatment of the case need not be here detailed. As soon as is practicable, the child should be allowed up in order to promote the expansion of the lungs. This may be helped by giving the child breathing exercises. In older children a series of Wood's bottles may be arranged, and the patient taught to blow his fluid from one into another. If the fluid in them is coloured, the child usually takes easily to this device, and gets quite attached to what he terms his "blow bottles." In younger children a trumpet may be used for the same purpose. Plenty of fresh air and good food should be allowed.

Where the temperature does not fall satisfactorily, the possibility of further collections of pus, in the pleura or in the pericardium, has to be considered.

Should the wound remain unhealed, the sinus may be scraped. Where this is unsuccessful, some operation to allow the chest-wall to fall in must be undertaken. In Estlander's operation, portions of several ribs are resected; in Shedd's, parts of the pleura and intercostal muscles are also removed. In both much deformity usually results.

PNEUMOCOCCAL HEART DISEASE.

With the exception of purulent pericarditis, pneumococcal infections of the heart are not common.

PNEUMOCOCCAL PERICARDITIS.

Purulent pericarditis due to the pneumococcus is much more common in children than in adults, and is particularly frequent in those under five years of age. Pericarditis may arise with the onset of pneumonia, when it is usually part of a general pneumococcal pyæmia; or it may develop at the close of the pulmonary infection in which case it is usually associated with an empyema. Often its origin seems clearly traceable to an undiscovered or ill-drained empyema.

Symptomatology.—The symptoms of pneumococcal pericarditis are in no way distinctive. Where it co-exists with active pneumonia, the symptoms are those of a violent pneumococcal pyæmia by which the patient is being rapidly killed. In the absence of active pneumonia, the symptoms are those of an unopened collection of pneumococcal pus, and are thus practically the same as those of a small, undrained empyema. There are prostration, fever (intermittent or remittent), sweating, wasting, with a polymuclear leucocytosis. Symptoms of cardiac failure are not marked, inasmuch as the pneumococcus produces comparatively little dilatation of the heart. Jaundice develops in a few cases, sufficiently often perhaps for its presence to give rise to a suspicion of pericarditis. Attacks of acute dyspnoea, with high fever and great prostration, are not uncommon, as Dr. Paget has noted. Such may occur in empyema, but are more frequent in lasting cases of purulent pericarditis.

The Physical Signs are seldom distinctive. A stage of pericardial friction is only very exceptionally present in purulent cases, and even then is usually very transient. As a general rule it may be laid down that in pneumococcal pericarditis effusion is present. The amount of pericardial effusion is rarely sufficient to give rise to characteristic signs. Enlargement of the area of the deep cardiac dullness to right and left is usually present; but of more importance is enlargement upwards. In exceptional instances the dullness may be confined upwards to under the left clavicle. In such cases the precordial dullness is of a resistant character and the heart's sounds are muffled; but such signs as these are the exception. There are usually signs of compression of the lower lobe of the left lung. Precordial oedema is a very rare sign of purulent pericarditis, but where present is of great value. A skiascopic view shows a large, clear shadow if the pericardium is greatly thickened. The pericardium may be palpated by the finger through an empyema walled, where this is present, and its condition thus rendered certain.

Morbid Anatomy.—In acute cases the surface of the pericardium are roughened and granular, and the sac contains yellow, fatty or slightly turbid fluid. In the more chronic cases, to which the term *pyopericardium* may be conveniently limited, the pericardium is much thickened, the apposed surfaces are covered with a shaggy deposit of fatty pyogenia, and the cavity is filled with thick, creamy pus. The size of the heart is not greatly increased, the myocardium suffering comparatively little. Endocarditic vegetations may be present, but such are not the rule.

Diagnosis.—In acute cases, where active pneumonia is present, purulent pericarditis cannot be diagnosed, except in some of the rare instances in which a true pericardial friction is heard.

In more chronic cases, which give rise to symptoms after the pneumonia has run its course, the diagnosis is still one of the utmost difficulty. So hard is it, that it is only by persistently being on the look-out for the condition, that there is the slightest chance of recognizing it. The diagnosis may be discussed under two headings, symptoms and physical signs.

The symptoms are usually suggestive of the presence of a collection of pneumococcal pus, but are of little value, as has been pointed out already, in localizing the pus to the pericardium. Indeed, since there are constantly signs of compression of the lower lobe of the left lung, in cases of *pyopericardium*, there is no little danger of suspecting some small, possibly interlobar, empyema on the left side; or should an empyema there have already been opened, a suspicion of a further unopened focus of pus in the left pleural cavity may well arise. On the other hand, it may be thought that the whole process is tuberculous, but an examination of the white cells of the blood as a rule will obviate this mistake.

The physical signs in the heart are often very misleading, and are seldom distinctive. A further enlargement of the deep cardiac dullness, to right and left after the pneumonia has run its course, is suggestive. In order that this may be of any use, we must of course have accurate measurements of the heart's dullness throughout the illness, while in the presence of an empyema in a small child it may be very difficult to map out the area of cardiac dullness. Of greater value is enlargement of the deep dullness upwards. Thus it well marked is a sign of very considerable value. Care must be taken that the dullness be misinterpreted and regarded as due to a tuberculous consolidation of the left upper lobe. In most cases, however, the amount of fluid in the pericardium is small, and there is no great enlargement of the deep dullness in an upward direction. Muffling of the cardiac sounds is also a sign of value; but this again is only exceptionally present in *pyopericardium*. Reddening and oedema of the skin of the precordium would be a very valuable sign if it were present, but it is of the utmost rarity; I have never met with it in a child.

The two most valuable signs remain to be mentioned. A diagram may be of the utmost help in a chronic case where there is much thickening of the pericardial wall. The only certain diagnostic sign is that of pulsation of the pericardium through an empyema wound. Should there be such a wound, this should be done in any doubtful case.

Failing these, where there is a grave suspicion of a pyopericardium, the pericardium may be explored under an anæsthetic. A small open operation is preferable to paracentesis.

The diagnosis of a pyopericardium is then a matter of the greatest difficulty. In spite of every care, a large number of cases escape recognition, and their possibility remains a bugbear to the clinician.

Course and Prognosis.—The acute cases which are part of a general pneumococcal infection developing with the onset of pneumonia, necessarily end fatally in nearly all cases; but the purulent pericarditis itself has probably little to do with causing death.

The more chronic cases, if untreated, are fatal. A spontaneous cure occasionally occurs in adults; but in children such a result must be infinitely rare. The condition may last for six weeks, and probably longer, but more often death takes place in a shorter time than this. It is almost always due to pneumococcal meningitis.

If diagnosed and drained, pneumococcal cases do extremely well, and scarcely any after-effects remain.

Treatment.—In the presence of active pneumonia, a purulent pericarditis in those rare cases in which it is then recognizable, is better left alone, and no attempt made to drain it at that time.

In the chronic cases, the pericardium should be opened and drained. The exact operation by which the pericardium is opened does not seem to matter. The anterior route appears to give quite satisfactory drainage, but should an empyema wound be present, the pericardium may be drained through it.

PNEUMOCOCCAL ENDOCARDITIS.

This is much less common than is the pericarditis due to the same organism. Like it, however, it is more frequent in children under the age of five years than in older patients.

It may be seen in two clinical groups of cases. In one it is associated with pneumonia, often with empyema and purulent pericarditis. The mitral and tricuspid valves are those usually attacked. They show small vegetations, whiter than the grey ones of rheumatism, which have to be distinguished from the irregularities seen normally on the edges of valves in small children.

The second class of case corresponds to a malignant endocarditis, often attacking valves already damaged by rheumatism. Both forms are quite uncommon in children.

PNEUMOCOCCAL MYOCARDITIS.

Myocarditis is not an important result of a pneumococcal infection. In an ordinary pneumonia there is comparatively little in the way of myocarditis. Where the pneumococcus has produced purulent pericarditis and endocarditis, myocarditis of a more severe grade may be present.

PNEUMOCOCCAL MENINGITIS.

Etiology.—In most cases, pneumococcal meningitis is associated with, and secondary to, pneumonia. In others it arises synchronously with the development of pneumonia. In a third group of cases the meningitis is secondary to empyema or peripneumonia, sometimes to middle-ear disease. Lastly, very rarely the condition is found without any demonstrable pneumococcal lesions elsewhere in the body, the so-called primary cases.

Pneumococcal meningitis is much more common in young children than in those over the age of five years.

Symptomatology.—Where the meningitis arises at the apex of pneumonia, the child may die very rapidly from the severe toxæmia, and no signs pointing to meningeal involvement—which is only very slight—may be present.

The symptoms of this form of meningitis are usually very severe. Repeated convulsions, general rigidity, tremor of the limbs, vomiting, and high fever are found. A slight squint is common. In infants, a bulging fontanelle is an important sign. Older children may give signs pointing to severe headache. Optic neuritis is not commonly seen, as the disease is so rapidly fatal. Towards the end of the case, some tendency to grouped breathing may be seen. Very occasionally hemiplegic weakness is present. The cerebrospinal fluid is increased in amount, under pressure, turbid, and showing an increase of albumin. Microscopically it is seen to contain an excess of polymorph leucocytes and pneumococci. Such cases as these terminate fatally within three to seven days.

Exceptionally, the disease may run a much less violent course. This is particularly seen where it is secondary to a chronic empyema or pyopneumothorax. Thus, for the last two or three days of life the child may become drowsy, a little rigid, occasionally convulsed, and dies in a state of coma. Post mortem, however, the entire surface of the brain and cord may be found covered with a purulent meningitis, perhaps an eighth of an inch in thickness in some parts. With such extensive meningitis and such quiet symptoms, it is impossible to escape the conclusion that the meninges have been involved for longer than the usual period of under a week.

Morbid Anatomy.—The meningitis is first seen over the superior surfaces of the frontal lobes. It is usually symmetrically distributed,

Where death occurs from the septicæmia rather than from the meningitis itself, a slight purulent exudate is found alongside the congested vessels on the vertex of the brain. In other cases the meningitis spreads over the frontal lobes and finally all over the surface of the brain (Fig. 26), and pus may also be found within the ventricles. In most cases the spinal ganglia are involved, the posterior surface showing the pusulent exudate most markedly. In universal cases care must be taken to exclude the possibility of a meningococcal meningitis by a bacteriological examination of the pus.

Diagnosis.—The most important signs of pneumococcal meningitis are repeated convulsions, bulging of the fontanelle in infants, and—the one quite distinctive sign—the presence of polymuclear leucocytes and pneumococci in the cerebrospinal fluid.

Many of the symptoms are simulated by pneumonia alone. These have been discussed on p. 95.

The differential diagnosis of the various forms of acute meningitis is given under the tuberculous type (Table 16, p. 127).

From otitis media, pneumococcal meningitis is often only differentiated with difficulty. Local signs in the ear, and symptoms suggesting pain in the ear, such as rolling or banging of the head, point to middle-ear disease; while bulging of the fontanelle or the presence of optic neuritis, are in favour of meningitis. Both conditions may co-exist. In cases of difficulty, only an examination of the cerebrospinal fluid is of avail in settling the diagnosis.

Intracranial abscess can usually be differentiated from pneumococcal meningitis by the presence of localizing signs and definite optic neuritis. The lumbar puncture again will decide the matter.

Prognosis.—It is extremely doubtful if recovery ever occurs from this form of meningitis. Death usually takes place within three to seven days; but as has been mentioned, there are reasons for thinking that exceptionally the disease may run a rather longer course. In the very acute cases it is probable that death is the result of the general toxæmia rather than due to the very early meningeal changes found post mortem in such instances.



FIG. 26.—PNEUMOCOCCAL MENINGITIS.
Showing purulent meningitis over most of cerebral cortex.

Treatment.—This consists in the use of sedatives, which are generally necessary. Nasal or esophageal feeding is usually required. The convulsions may be relieved by the administration of chloral or by similar puncture.

PNEUMOCOCCAL OTITIS MEDIA.

This is the most frequent form of acute middle-ear disease in children, although other pyogenic infections are common. In infants, a few cases are tuberculous.

It may be well to mention here, that in autopsies on children under the age of two years it is an almost invariable rule to find mucus in the middle ear. Commonly there is no associated injection of the tympanic membrane. Such a condition as this may be found equally in cases dying with high fever, general rigidity and convulsions, and in those in which no such symptoms are present. For these reasons, such cases in infancy should not be regarded as instances of otitis media.

Etiology.—Otitis media frequently occurs during or following an attack of pneumonia. Seeing that the pneumococcus is a constant inhabitant of the nasopharynx, otitis media due to this organism frequently exists without any other foci of infection. It is particularly prone to develop in children with enlarged tonsils and adenoid vegetation.

Symptomatology.—We are here only concerned with the medical features of the disease as it occurs in children. In some cases the chief symptom is that of fever, and otitis media has to be remembered as a cause of unexplained pyrexia in a child. In others, there are severe symptoms, drowsiness, rigidity, head-rotation, and squinting; all such symptoms, that is to say, which may be found in pneumonia or in acute meningitis. In addition, there may be signs that there is severe pain in the ear. Where this is not actually complained of, it may be shown by the child's hand being put constantly to the side of the head, or by rolling or banging movements of the head. Redness and bulging of the tympanic membrane may be present. Optic neuritis is present in exceptional cases. The cerebrospinal fluid is normal.

Diagnosis.—This may be a matter of considerable difficulty. Mention has already been made of the possibility of otitis media being a cause of an unexplained attack of high fever in a child. Where pneumonia co-exists, in the absence of the symptoms suggestive of pain in the ear or of local signs of inflammation, the condition can hardly be diagnosed, as pneumonia itself may give rise to all the other symptoms of middle-ear disease. The absence of injection and bulging of the membrane does not exclude the possibility of otitis media.

Again, from acute meningitis, usually from the pneumococcal type, the differentiation of otitis media may be very difficult. In the absence of signs or symptoms pointing to the involvement of the ear, bulging of the fontanelle, or the presence of optic neuritis, will be in favour of meningitis. The most satisfactory method of a differential diagnosis is by means of an examination of the cerebro-spinal fluid. If thought advisable, the membranes of the ear, although appearing normal, may be punctured under an anæsthetic. This should, if the ears alone be at fault, relieve the symptoms.

Treatment.—The tympanic membrane should be punctured under a general anæsthetic. As, in a small infant this is not an easy procedure, relief to the symptoms until special aid can be obtained may be given by the application of a leech behind the ear. Where the pus escapes spontaneously or as the result of a puncture the ear should be syringed out with some warm lotion and kept covered with a dressing.

PNEUMOCOCCAL PERITONITIS.

Etiology.—This is not a common condition, but it occurs with sufficient frequency to constitute a source of danger. It is more common in children, and particularly in young children, than adults. Girls are more often affected than boys.

In many cases it arises as part of a generalized infection with the onset of pneumonia. In others it develops towards the termination of a pneumonia, or as secondary to an erysipelas or pyopericardium. Very occasionally it exists as a "primary" condition, no other pneumococcal lesions being demonstrable.

Symptomatology.—In many cases the symptoms and signs of pneumococcal peritonitis are indefinite. Where it exists as part of a general pyæmic infection, the symptoms referable to the abdominal condition are very slight, and are often overlooked entirely. As a rule, the most prominent symptoms are abdominal distention, with diarrhoea or constipation, some limitation of movement, and tenderness and rigidity of the abdominal walls. These last signs, however, are not nearly so definite as in peritonitis secondary to a perforated viscus. No free fluid is present at first, but a small amount develops within about twenty-four hours. In other cases, the abdominal symptoms are more marked, and the condition resembles that of an "acute abdomen." In such instances, toxic symptoms are comparatively slight, and, very rarely, may be absent altogether. In a few of these cases the peritoneal inflammation is shut off by adhesions, usually in the hypogastrium or pelvis, but in the majority the peritonitis is generalized.

Morbid Anatomy.—Usually the peritoneum is universally inflamed, the interstices between the coils of distended intestine, are filled up by thick pro-lymph, and a small amount of free turbid fluid is present. Occasionally, the upper part of the abdomen only is involved, although no gross free adhesions are present. Such cases appear to be due to a spread of infection through the diaphragm, death having occurred before the peritonitis has become general. Local collections of pneumococcal pus without general peritonitis, are found in some instances.

Diagnosis.—Where there is co-existent active pneumonia, this is a matter of the greatest difficulty. Reliance has to be placed upon increasing distention, decreasing mobility of the abdominal walls, the tenderness and rigidity. All these, however, are but slender guides, particularly where the pneumonic symptoms are severe, and might legitimately be accompanied by abdominal symptoms (e. g. see without peritonitis). Where the child survives for a time, the development of free fluid in the abdomen is a sign of great value, especially if, with the increase of the abdominal and general symptoms, the pulmonary condition shows signs of resolution.

Where acute abdominal symptoms are marked, the diagnosis is seldom made until the abdomen is opened; frequently, indeed, the infecting organism is not suspected until a bacteriological examination reveals the pneumococcus. In such cases, however, the evidence is sufficient to lead to laparotomy, which is of course the important matter.

Prognosis.—In the cases which are part of a general pneumonic infection, the outlook is almost necessarily fatal. In other cases, however, where the diagnosis can be made and surgical treatment adopted, the prognosis is much better, and many recoveries occur. Of spontaneous recoveries of general pneumococcal peritonitis in children, very little is known.

Treatment.—This is on the same lines as that for pneumococcal peritonitis. Many cases in which a severe pneumonia, pneumonitis, and various pneumococcal metastases are present, are not amenable to treatment. In other cases the abdomen should be opened and drained as soon as the diagnosis can be suspected with any degree of assurance.

PNEUMOCOCCAL NEPHRITIS.

Hematuria occasionally occurs in cases of pneumonia, the urine showing blood, casts and pneumococci. Should the child survive the pneumonia, the urine usually rapidly becomes normal, and no special treatment is required.

On the other hand, some cases of acute nephritis with much diminished urine, hæmaturia and œdema, show pneumonia, while still

more are associated with bronchitis. It is more than probable that a certain proportion of these are pneumococcal in origin, the kidneys bearing the brunt of the attack.

PNEUMOCOCCAL ARTHRITIS.

Pneumococcal arthritis, although an uncommon condition, is by far the most frequent form of infective arthritis in infancy, and up to the age of five years. About half the cases show an antecedent pulmonary affection, while others may be associated with pneumococcal otitis media. In some the arthritis appears to be "primary."

The joints of the lower extremities, the hips and knees, are most often affected than those of the arms. More than one joint may be involved. Together with febrile constitutional symptoms, the joint is swollen and tender, and in severe cases the skin may be both shiny and reddened. The affected limb is held stiff, but is not extremely painful. The pus contained in the joint is thick and creamy, while in the more severe cases it is thin and watery.

Some cases of acute osteomyelitis are due to the pneumococcus; but so far as I have seen these, the neighbouring joint is always involved.

Prognosis.—This is grave. The death-rate is given as 75 per cent in infants under one year, 24 per cent in those between one and two years, and 50 per cent during the remaining years of childhood (Nitch). Death is due to other pneumococcal manifestations, and in the absence of these the condition is not a fatal one.

Treatment should consist as a rule of opening and irrigating the joint.

PNEUMOCOCCAL ABSCESSSES.

These may occur during or following an attack of pneumonia. They may be formed in the lungs, brain or subcutaneous tissues. A rare type is when the mediastinal glands or nodes suppurate. In such a case an abscess may track round the chest-wall, between the pleura and the ribs. Some cellulitis may cause an extra-pleural abscess along the course of the ribs, without any suppuration in the mediastinum. These are rarely of any clinical interest.

PNEUMOCOCCAL CONJUNCTIVITIS.

Conjunctivitis of pneumococcal origin has been lately recognised, and is said to be, after the gonococcal, the commonest form of ophthalmia neonatorum.

OTHER PNEUMOCOCCAL INFECTIONS.

The pneumococcus is probably responsible in part or wholly for some cases of meningitis. Instances of gastritis and enteritis, particularly colitis, have been recorded as due to this organism. Pneumococcal cystitis occurs occasionally in children as in adults.

Such conditions show no clinical characteristics of sufficient interest to necessitate more than the mention of their occurrence.

II.—THE TUBERCULOUS INFECTION.

When it has been stated that the statistics of various authorities show that of the deaths occurring among the children of the London poor nearly one-third are due to tuberculosis, it becomes unnecessary to emphasise further the appalling frequency and fatality of the infection among children.

ETIOLOGICAL FACTORS.

Age-incidence.—In dealing with the age-incidence of a disease so insidious in onset as is tuberculosis, the only reliable figures are those obtained from post-mortem records. By the study of these, however, important facts are brought out, but it is to be remembered that the younger the patient the more likely is the infection to become generalised, and thus fatal.

Tuberculosis, in common with many other infections, is more frequent in children during the first five years of life than later. In Dr. Still's figures, practically four-fifths of the total cases of tuberculosis in children under the age of twelve years are seen to occur during the first five years of life.

Death from tuberculosis in the first three months after birth is very rare, and up to the age of six months is exceptional. During the second six months, and especially from the ninth month onwards, it becomes much more frequent. It is during the second year that the mortality of the disease reaches its maximum, and within this period no less than 25 per cent of the deaths from tuberculosis in children occur. In each of the third and fourth years, the mortality is about one-half that of the second year; while in the fifth it becomes again much reduced. From this time onwards the number of deaths from this disease shows a rapid and progressive diminution.

Modes of Infection.—From the point of view of prophylaxis, all information concerning the modes of infection is of value. Unfortunately, however, much that appears of primary importance to this subject cannot yet be regarded as settled.

We have to distinguish clearly between the facts that are definitely ascertained and the theories that have been advanced to explain them.

The age-incidence of tuberculosis has already been stated. That deaths from the disease are most common between the twelfth and twenty-fourth months, and that the infection begins to be fairly frequent at about the ninth month, are definite facts. Secondly, the results of pathological investigation have been shown to be nearly constant by various observers. These are to the effect that primary tuberculous lesions are found most frequently to occur in the lungs and bronchial glands in connection with the respiratory system, and in the intestine and mesenteric glands in connection with the alimentary system. The relative frequency of these two groups is a matter of some moment. No one who has done many post-mortem examinations in children can fail to be struck by the fact that evidence pointing to a primary thoracic infection is far more commonly found than is evidence of a primary abdominal infection. All statistics prove this. Various observers have found that the abdominal cases are outnumbered two, three, or even more times by the thoracic. Dr. Still has further shown that in fatal cases during the milk-feeding years (under one year and under two years), the proportion of thoracic to abdominal cases is not reduced, but rather increased.

Again, it is unfortunately beyond doubt that London milk not very rarely contains tubercle bacilli; and further, that tuberculosis can be experimentally produced by feeding animals on infected material.

There come then the important questions: Why are children so prone to tuberculosis; and, how do they become infected? The answers to them cannot at present be definitely given.

We know that children have but a poor immunity to many infections, and that, if acquired, the infection tends to become generalized. We know further that a tendency to pulmonary disease, bronchitis, and pneumonia, is very marked in early life. Is there any added factor? Many have held that an infected milk supply is the cause of the age-incidence of tuberculosis. If, however, it is held that thoracic tuberculosis is the result of infection by inhalation, while abdominal disease is the result of a swallowed infection, such a view becomes untenable, as the abdominal cases are in a marked minority. There is, however, experimental evidence to the effect that the bronchial glands alone may become infected from material ingested as food, and thus it is probable that our ideas on these points require some rearrangement. It is by no means easy, for instance, to imagine that the direct inhalation of tubercle bacilli into the lungs is a very common event, for it would seem so much more likely that even air-borne bacilli would become adherent to the moist lining of the nostrils and pharynx, and thus be swallowed rather than inhaled. Experimental evidence is at present far from clear, different investigators having obtained contradictory results. Therefore we cannot yet say more than that it is quite possible that the separation between the methods

of infection in throat, and abdominal tuberculosis is not so clear cut as has been supposed. Infection via the tonsils or ears may occur, but is uncommon.

Certain other points require consideration. That tuberculosis should become common just at the age when children are learning to crawl and walk seems too great a coincidence to be neglected. It appears extremely likely that at such an age a child would be particularly prone to infection from contaminated dust and dirt, especially when it is remembered how it puts its fingers and all treasure trove into its mouth. Again, the habit that the hospital mother has of putting the baby's comforter in her own mouth before giving it to the child is fraught with considerable danger. The same thing is done to the best of a baby's bottle. Nor does the breast-fed child escape this danger, for it is a common practice for a mother to masticate the nipple at her breast with her own saliva before the baby takes it in its mouth. In many other ways infection may be conveyed to the child; indeed, the baby in its helplessness runs a greater danger of infection than the older children, in a family where the mother is tuberculous.

Predisposing Causes.—Evident illnesses are well known to exert an influence in predisposing towards tuberculosis. Of these the chief are measles and whooping-cough. Possibly the age-incidence of tuberculosis is explained to some extent by the influence of these diseases. They probably act both by depressing the general vitality and resistance of the child, and by setting up a catarrh of the lungs with secondary inflammatory changes in the bronchial glands.

Faulty hygienic conditions as predisposing causes of tuberculosis hardly require emphasis at the present time. It is in children that the worst results are seen of over-crowding, bad ventilation, poor food, lack of fresh air and sunshine.

GENERAL TREATMENT.

Prophylactic Treatment is a matter of the greatest importance. From what has been said above, we must recognize that the danger of infection comes chiefly from three sources; namely, an infected person, infected dust, and infected milk. Of the relative frequency of infection from these causes we cannot as yet speak with certainty. The avoidance of the first two sources is to be attained by those methods which many "health societies" are now doing such good work in popularizing. It only remains to say that where children under five years of age are in question, every possible care must be taken, for even the smallest risk is a great danger at this age. The possibility of milk-infection renders it essential that all milk given to children should be sterilized by scalding, pasteurization, or boiling.

General Remedial Treatment.—Unfortunately this is often out of the question with children. Wherever possible, however, it is to be carried out on the same lines as for adults, remembering that as the resistance to the infection is less in young than in older subjects, treatment must be both more prompt and strict.

Fresh air is the first requisite. A change of climate is usually necessary. Children do well in most localities which are suitable for adult consumptives, but as a rule the warmer health-resorts, especially those at the seaside, are the most satisfactory for them. In summer time the various east-coast places are very suitable, e.g. Margate, Broadstairs, Cromer, etc. In the winter the south coast is preferable, Bournemouth, Torquay, or the south Cornish coast. In spring or during a mild winter, Ramsgate, Folkestone, or Westcliff are excellent. If an inland resort is preferred, a child may be taken on to the hilly districts, such as Dartmoor, the air of which is practically that of the seaside, Hindhead, or the Cotswold or other hills. On the Continent there are many places highly recommended for tuberculous children.

The diet must be the lightest which can be taken without causing indigestion. Milk is the most valuable of all foods for tuberculous children. Cream and raw meat juice are likewise very useful. The former can be used in place of cod-liver oil if necessary. Eggs, milk-puddings, minced meat and gravy should be given. In the presence of diarrhoea fats should not be given (p. 142).

Amongst the drugs of use in the treatment of tuberculosis, cod-liver oil takes the first place. It is as a rule most easily taken in combination with extract of malt, or as an emulsion with hypophosphites. Scott's preparation is of particular value, the emulsification of the oil being so successfully carried out. Other children will take cod-liver oil better if considerably sweetened with glycerin, as in a mixture of equal parts of oil, lime-water, and glycerin. Cod-liver oil should not be given where there are signs of a deranged digestive tract, but loss of appetite alone is not a contraindication to its use. Where the oil cannot be taken, extract of malt may be given alone.

Other drugs may be of service. Creosote, taken internally or in the form of inhalations, seems certainly of value. The methods of administration are dealt with later (pp. 144 and 145). Iron, in the form of steel wine, the syrup of the iodide or phosphate of iron, may be used for combating the anaemia. The iodide of iron seems of particular service in cases of tuberculous adenitis. Tonics for the improvement of the appetite are sometimes useful. Tuberculin injections are being used, but as yet the reports on the value of this method in children are incomplete. For them, the administration of tuberculin is unusual saline by mouth has much to recommend it in place of the hypodermic method. Such a dose is best given an hour before breakfast.

In the treatment of tuberculosis, drugs are secondary to climatic measures of importance.

GENERAL MILIARY TUBERCULOSIS.

Miliary tuberculosis of the meninges, thoracic and abdominal organs may show itself in various clinical forms. It may perhaps be of use to mention the chief of them.

1. A very common form is that in which *tuberculosis meningitis* is alone recognizable clinically, nothing abnormal being demonstrable in the chest or abdomen. This is an important type of general tuberculosis, because it is usually found in fat infants, a fact not seldom a cause of difficulty to practitioners. It is unfortunately true that a well-nourished infant may have and may be dying of tuberculosis. The reason is clearly seen at an autopsy on such a case. As a rule, the only deposit of caseous tuberculous material is found in the glands at the bifurcation of the trachea, and from this the fatal meningitis has arisen. The lungs and pleura show sparsely-scattered tubercles, the spleen may be slightly enlarged, and on it, and on the liver and kidneys, on a few superficial tubercles. The peritoneum under the diaphragm and in the flanks may likewise show a small number of tubercles. The intestines as a rule are not ulcerated, and the mesenteric glands may be normal or show a very early infection. Obviously, the glandular infection was primary, and on dissemination occurring, death is caused by the meningitis before much wasting takes place.

2. Quite different is another group, also found most commonly in infants, in which the case is looked upon as one of *simple marasmus*. Here wasting is the marked feature, and the true diagnosis may not be suspected until the autopsy, although possibly enlargement of the spleen may have given rise to a suspicion of tuberculosis. In a wasted, scolic infant, the signs of meningitis may be very indefinite, or may be regarded as merely the terminal symptoms of a case of chronic wasting. Yet pathologically, in addition to the meningitis, all the organs are found to be severely infected, to show not only acute miliary tuberculosis, but a good many caseous lesions. In such cases as these, tuberculous masses in the brain are not uncommonly found.

The diagnosis of this type of general miliary tuberculosis is only to be arrived at by most careful examinations for signs in the chest or abdomen being made at intervals throughout the illness. Progressive emaciation, in the absence of diarrhea or vomiting, with enlargement of the spleen, is the suggestive picture. Bulging of the anterior fontanelle is not often seen with the onset of the meningitis in such emaciated infants.

3. A third type of the disease may be seen in older children, and is characterized by a protracted fever, so that the case resembles one of enteric fever. The similarity is shown by the pyrexia, unconsciousness, delirium, bristling sounds in the lungs, and enlargement of the spleen. The differential diagnosis is discussed under typhoid fever (p. 205).

4. The last group of general miliary tuberculosis that need be mentioned, is that in which there is recognizable disease in the chest or abdomen. Generalization may occur as a terminal lighting-up of a chronic infection or in connection with acute tuberculosis of some organ. The most difficult cases for diagnosis are those associated with acute pulmonary disease, the tuberculous nature of which is hard to recognize.

TUBERCULOSIS OF THE NERVOUS SYSTEM.

For the most part this consists of acute meningitis. Mention must also be made of intracranial tuberculous tumours. The spastic paraplegia secondary to tuberculous spinal caries, is dealt with in surgical text-books.

TUBERCULOUS MENINGITIS.

By this term is usually meant a general miliary tuberculosis of the cerebrospinal meninges. Localized tuberculous meningitis may no doubt be present in association with tuberculous masses in the brain; but as a rule tuberculous meningitis means a generalized and not a localized infection. It is important to bear this in mind in relation to reported recoveries of cases of "tuberculous meningitis."

Etiology.—The age-incidence of tuberculous meningitis has already been given on page 186. Injury to the head certainly seems to predispose to the onset of the disease just as injury to a joint predisposes to tuberculous arthritis.

Tuberculous meningitis is invariably secondary to some focus of infection elsewhere. This is most commonly to be found in the mediastinal glands; less often in the mesenteric glands. The amount of tuberculosis in the body may be extremely small, limited just to the glands at the bifurcation of the trachea, and thus, as has already been emphasized, meningeal tuberculosis may be found in children who are well-nourished and who show no clinical signs of tuberculosis in the chest or abdomen. Occasionally it follows operations upon tuberculous cervical glands, or may be secondary to disease of the ears or bones.

Morbid Anatomy.—At autopsy, tuberculous meningitis is invariably found to be cerebrospinal in distribution.

In the brain, the infection is seen at its earliest at the anterior base (in the region behind the optic chiasma) and in the Sylvian fissures. In the latter positions the meningitis is often the most obvious, running together the lips of the fissures and sometimes interfering with the patency of the blood-vessels there. From the anterior base, diffuse miliary tubercles may usually be found spreading backwards towards the cerebellum and upwards along the vessels over the lateral

apex of the cerebellum. A slight degree of internal hydrocephalus is generally present. In some cases, tuberculous masses (generally multiple) may be found in the brain substance, usually connected with the meningeal vessels.

On the spinal cord, the meningitis is most marked on the posterior surface. It is, as a rule, best seen between the posterior nerve roots and on the inner aspect of the dura mater. This surface of the dura loses some of its glossiness and appears granular. Examination by means of a lens is often advisable.

Symptomatology.—Tuberculous meningitis shows some differences in its symptoms according to the age of the patient. In young infants and in older children certain peculiarities in the disease may often be found. These will be referred to later.

The disease, as it occurs at the usual age, is often described in three stages,—prodromal, irritative and paralytic. Such a division is, however, of little clinical value.

The onset of tuberculous meningitis is as a rule insidious, and it is here that the main difficulties in diagnosis arise.

The earliest symptoms which are most constantly present are headache, vomiting, constipation, and a change in disposition.

Headache, rarely absent altogether, may be quite slight, or may be only spasmodic. Very severe headache is unusual except in the case of older children of seven or more years. Vomiting is again rarely entirely absent. Most usually it is present for a few days, and as the child becomes worse it passes off. It may be of the cerebral type, explosive, not preceded by nausea, and bearing no relation to food; but even this is not the case. Frequently, indeed, the initial symptoms of tuberculous meningitis are regarded simply as a "bilious attack." In other cases, the vomiting increases in severity, and as the child becomes drowsy and the eyes sunken, a mistaken diagnosis of cyclical vomiting may be made as the breath and urine are both loaded with acetone, the result of the starvation which the child is undergoing (p. 86). Constipation is the rule in the early stages of the disease, but diarrhoea may sometimes be found, depending often but not invariably, upon severe intestinal irritation. A change in disposition is always present, but may not be at once suggestive of the disease which is commencing. Most characteristically there develop drowsiness and irritability. When the child is left undisturbed, it is noted to be unusually quiet, so far as to take notice of its toys, and when attended to, it appears to resent interference, to cry and to wish to be left alone. As the disease progresses, the drowsiness, in spite perhaps of occasional partial remissions, gradually increases. In young infants these signs are as a rule all that are observable in the way of an alteration in disposition. In older children, more confusing symptoms may arise, such as acute mania or some mental perversion which may be regarded as hysterical. The movements of the limbs

may be tremulous. Soon, however, the drowsiness manifests itself. The child when undisturbed lies quietly, often curled up in bed; he refuses to open his eyes, for photophobia is often present, and when disturbed is very fretful: indeed, the picture becomes typically one of cerebral irritation.

In some such way as this the majority of cases of tuberculous meningitis develop. At the end of about a week the patient is so comatose that he can be roused with difficulty if at all; he lies curled up in bed, resents interference, the vomiting is passing off and generalized rigidity is present. This is shown at its earliest by pain being produced on forcible flexion of the head, or on attempts being made to elicit Kernig's sign (p. 15). At about this period convulsions commonly occur and a squint develops; the disease is now easily recognizable.

Less commonly the onset of the disease is much more abrupt, and a convulsion may be almost the first symptom. Much more rarely some paralytic symptom, spasm, palsy, or hemiplegia is the first symptom calling attention to the child's ill-health. Two types of hemiplegia are seen in connection with tuberculous meningitis. The first is of sudden onset, and is unassociated with any signs of meningitis for a time. In three such cases observed by the author, general meningeal symptoms did not develop for two, four, and six weeks after the onset of the hemiplegia. The paralysis is probably the result of an embolus of tuberculous material, or possibly is due to sudden changes occurring in the neighbourhood of a latent tuberculous cerebral tumour. It is probable that some of these cases may recover, but it is difficult to prove this. In the second type of case, the hemiplegia is of gradual onset, and is associated with other early signs of meningitis. It is in fact due to tuberculous meningitis actually present and interfering with the flow of blood in the vessels in one Sylvian fissure.

The disease as it progresses becomes very characteristic and easily recognizable at sight. The child gradually becomes more and more deeply comatose, its irritability on being disturbed lessening. The generalized rigidity increases. Slight head-retraction may be present, but this is practically never a marked feature. Swinging movements of the eyes are observable, and a squint is usually present. The abdomen tends to become retracted in the absence of abdominal tuberculous of any severity. The tachy-pneural is present. Optic neuritis is by this time usually definitely present in slight degree, and choroidal tubercles may be found in the fundi of the eyes. The arms are often extended, and may be over-extended, the wrists and fingers flexed. Syncopal attacks are not infrequent. Difficulty in swallowing has by now been replaced by total inability, and feeding by means of a tube is necessary. Catheterization may be required as the unconsciousness deepens.

The last stage is but an acronutation of the preceding. The child lies on its back (so little is the head-retraction) and is deeply comatose.

The face is flushed, sweating and becoming cyanosed. The eyes are open, and covered with a filmy deposit of mucus. The pupils are dilated and possibly unequal. There is froth on the lips. The breathing is rhythmic, periods of apnea alternating with groups of stertorous respirations (cerebral breathing). With some terminal convulsions and a rapidly ascending temperature, death occurs.

The cerebrospinal fluid as withdrawn by lumbar puncture in tuberculous meningitis is under abnormal pressure. It is clear or, at the most, opalescent, containing some flakes of lymph. It shows when boiled a heavy trace of albumin, and its action in reducing Fehling's solution is lowered or absent. There are many cells present in it, the great majority of which (80 to 90 per cent) are lymphocytes. Rarely, polymorphonuclear cells are as numerous as the lymphocytes. In addition, tubercle bacilli are present, and may in a large proportion of the cases be found by examining microscopically the fine sediment or clot which forms when the fluid has been allowed to stand for twelve hours.

The temperature and the pulse- and respiration-rates require special mention. The amount of fever present is extremely variable, and depends more upon the extent of tuberculosis present elsewhere in the body rather than upon the meningitis itself. Where the lungs are severely affected, the temperature is likely to be high and fairly sustained. Most commonly in tuberculous meningitis, the temperature is not greatly raised until towards the end, and runs irregularly at 99° to 101°. The fever increases in amount during the last two or three days, and at death hyperpæxia is often seen. The pulse is at first rather quickened, and tends to be irregular, but this disturbance of rhythm is not a sign of much diagnostic value in a sick child. With the onset of stupor, the pulse is prone to become perceptibly slowed, falling in rate to 60 or even to 40 per minute. During the last stages, the pulse-rate again increases, and towards death the pulse becomes very rapid, feeble, and often uncountable. The respiration-rate, in the absence of extensive disease of the lungs, may become lowered with the slowing of the pulse-rate. Other changes are perhaps of greater interest. Very early in the disease perpetual yawning may be present, and be a sign very suggestive of tuberculous meningitis. Later, in place of the yawns, are occasional deep sighing respirations. As the disease progresses, the breathing begins to get rhythmic or grouped, until finally true "cerebral breathing" is seen, in which the respirations (often of equal depth) are grouped together, each cycle being separated from the next by a period of apnea (p. 10). Even when fully developed, such breathing is not necessarily a sign of immediate death, for it may exceptionally persist for as long as a week.

Lastly, there may be recognizable signs of tuberculosis in the cervical glands, chest, or abdomen. Enlargement of the spleen is perhaps the most constant of such suggestive signs.

Tuberculous meningitis, as it occurs in infancy before the closure

of the anterior fontanelle requires short mention. Here the disease is very rapid, and is not accompanied by much pain. As a rule, after a very short time of restlessness the drowsiness has developed so greatly that the patient becomes unconscious. There is bulging of the anterior fontanelle, a sign of very great diagnostic value as the absence of screaming or convulsions. Optic neuritis is not so well seen in these cases as in those in later years; indeed, it is exceptional here while the rule in older children. Very rarely there occurs a form of tuberculous meningitis in which the lesions are situated at the posterior base of the brain. In such a case the signs will be exactly those of the ordinary form of posterior base meningitis of meningeococcal origin and the examination of the cerebrospinal fluid will alone give a clue to the correct nature of the disease.

As it occurs in older children of an age of seven years or more, tuberculous meningitis is a truly distressing disease, the symptoms resembling those of the same disease in adults. The pains in the head, neck and back are of a most severe type. The "hydrocephalus cys.," a sudden shrill shriek, may occur, but it is a sign more often described than heard. The comatose stage does not develop rapidly, and for many days the child may have to be kept under morphia for the relief of his pain.

Diagnosis.—In making a diagnosis of tuberculous meningitis, we are as a matter of fact giving an opinion on three points, namely, that there is organic intracranial disease; that this is of the nature of an acute meningitis; and that it is of tuberculous origin. It is well, perhaps, to deal with the diagnosis under these three headings.

1. Tuberculous meningitis may be closely simulated, especially in its earlier stages, by many conditions in which there is no organic intracranial disease. Allusion has already been made to the fact that the condition may be overlooked and the symptoms attributed to a "bilious attack" or hysteria, or the illness may be called cyclical vomiting, where the vomiting is very severe and acetonaemia present. It is no other way, cyclical vomiting may be excluded by giving large doses of alkali to the child. If the urine can be rendered alkaline in this way, and yet the symptoms do not cease, they cannot be due entirely to acid intoxication.

Three mistakes are made, however, in connection with various infections in which symptoms closely simulating tuberculous meningitis may be present without any gross intracranial disease (meningismus). The most common of these are acute pneumonia, otitis media, enteric fever, and influenza. These may themselves cause unconsciousness, convulsions, generalized rigidity, slight head-retraction, Kernig's sign, squints, tachycerebrak, and even the slightest suggestions of rhythmic breathing. The differential diagnoses are given elsewhere in this book, and need only here be summarized. A bulging anterior fontanelle or optic neuritis will point to raised intracranial pressure,

While chloroform tubercles, or the results of the examination of the cerebrospinal fluid, may point definitely to tuberculous meningitis. As has been emphasized in dealing with acute pneumonia (p. 95), it is unwise to make a diagnosis of meningitis at a time when there are definite symptoms or signs of pneumonia. In the absence of pulmonary signs the symptoms of pneumonia are usually distinctive: the flushed face, the heightened respiratory rate, the grunting expirations, and the cough, are very characteristic.

2. If then we can be sure that the child is suffering from an acute illness in which there is a raised intracranial pressure, we have only one condition to consider apart from acute meningitis—namely, intracranial abscess. This may often be distinguished by its association with disease of the ear; but in some cases it is practically impossible to make a diagnosis except by examination of the cerebrospinal fluid. In intracranial abscess this is normal, in acute meningitis abnormal. Intracranial abscess is of rare occurrence under the age of five years.

3. Lastly, as to the form of the acute meningitis. The common forms are those due to the pneumococcus or allied organisms, to the meningococcus (usually epidemic or posterior base meningitis), and to the tubercle bacillus.

The first is as a rule easily distinguishable from tuberculous meningitis if any form of meningitis be suspected, by its close association with pneumonia or erysipelas, its very rapid course, the absence of the characteristic signs of tuberculous meningitis, and by the examination of the cerebrospinal fluid.

The usual differences between tuberculous and posterior base meningitis are set forth in Table 19.

Prognosis.—Acute tuberculous meningitis of the usual type—that is a secondary and cerebrospinal infection—is a disease which causes death within three weeks as a rule. The onset of rhythmic breathing and a rapid ascent of the temperature usually foreshadow the occurrence of death within two or three days.

The question arises, Does a case of tuberculous meningitis ever recover? Dr. A. E. Martin has recently investigated this matter (*Brain*, 1905), and finds that twenty undoubted cases of recovery have been reported since 1894. Of these only about half were in children under twelve years of age. In them the cerebrospinal fluid was either found to contain tubercle bacilli or to produce tuberculous when injected in guinea-pigs. In most a fatal relapse occurred within a few months. Where an autopsy was made at a subsequent date, the old lesions found were for the most part localized, and sometimes in atypical positions.

We must then allow that on very rare occasions tuberculous meningitis may exist without causing death. That a general cerebrospinal meningitis, such as is the rule in tuberculous cases, ever recovers, is almost unthinkable.

Treatment.—Before unconsciousness supervenes, sedatives are as a rule necessary. In older children, in whom there is much pain, morphia should be administered hypodermically. As the child becomes comatose it will need to be fed by means of a tube. Care must be taken to prevent the bladder becoming over-distended. Sedative drugs may be employed to allay the twitching movements and groaning that may occur at this stage. It should be explained to those round the bedside that these distressing movements and sounds are not signs of pain, and that the child is quite unconscious.

	TUBERCULOUS MENINGITIS	
	First Year	Second Year
Onset of age	First year	Second year
Eyes	Icterus No fundal changes Spasmodic staring Squints, late	Photophobia Optic neuritis Chorioid tubercles Squints, early
Mouth	'Champing' movements	No movements
Tachicardia	Slight or absent	Marked
Head retraction	Marked	Absent or slight
Other organs	Initial bronchitis or pneumonia	Spleen often enlarged; possibly other signs of tuberculosis
Cerebrospinal fluid	Turbid Polyuclear cells Micrococci	Clear or flaky Mainly lymphocytes Tubercle bacilli

Table 10.—*Menigeo-encephalitis* (TUBERCULOUS MENINGITIS). FROM THE BRITISH MEDICAL JOURNAL.

Convulsions may be relieved and to some extent prevented, by the use of bromide or chloral which may be given with the nasal feeds or per rectum. They may also be checked by means of lumbar puncture, but care should be taken lest by this method a child only recently become unconscious should be revived sufficiently to allow it again to feel pain.

The vomiting, if troublesome, is best relieved by means of alkalis, with gastric lavage if necessary. The bowels may be kept open by small doses of calomel or by injections of glycerin.

TUBERCULOUS INTRACRANIAL TUMOURS.

Tuberculous masses in the brain, if they give rise to any symptoms at all, as a rule cause those of an intracranial growth, and as such these tumours are considered elsewhere. It is to be remembered

that they are often entirely latent, frequently multiple, and that they are the commonest type of cerebellar tumour in a child.

In the case of a deep-seated tuberculous tumour, the cerebrospinal fluid remains unchanged. But where the deposit is superficial the fluid shows a considerable number of lymphocyte cells. Tubercle bacilli are rarely to be found in it.

It is probable, however, that tuberculous tumours may give rise to symptoms other than those of intracranial growth, namely those of tuberculous meningitis. The presence of tuberculous masses may be surmised in a case of tuberculous meningitis, when in the early stages of the disease optic neuritis is well-marked. Probably some of the cases of tuberculous meningitis which are recorded as having recovered, are cases in which there has been a caseous mass in the brain associated with some localized meningitis. Although recovery is not likely to come even in such cases as these, yet they are less liable to be fatal than is a generalized meningeal infection.

CHOROIDAL TUBERCLES.

In a large number, probably a majority, of cases of acute military meningitis, choroidal tubercles are present. They are seen in the periphery of the eye-grounds as small yellowish-white oval or round areas in close relationship to the blood-vessels. They have to be distinguished from patches of oedematous exudation and from the atrophic areas of syphilitic chorioiditis.

Although present in many cases of tuberculous meningitis, they are very seldom of any diagnostic value, as by the time of their appearance the nature of the disease is clearly manifest. Their presence, however, is for practical purposes proof of an acute military infection of the meninges.

TUBERCULOSIS OF THE RESPIRATORY SYSTEM.

TUBERCULOSIS OF THE TONSILS.

This is an extremely uncommon condition in childhood. Whatever part the tonsils may play in predisposing towards tuberculous otitis, it is certainly very rare to find microscopic evidence of tuberculosis in the tonsils themselves.

Retropharyngeal abscess is dealt with on p. 331. Tuberculosis may produce the condition in two ways: by the softening of mucous retropharyngeal glands, and by tuberculous caries of the cervical vertebrae.

TUBERCULOUS LARYNGITIS.

This is a disease of little importance in children. It is occasionally seen in autopsies on infants who have died of general tuberculosis: but at this age is rarely of clinical interest, producing only slight hoarseness. In older children, tuberculous laryngitis is less rare and

differs in no way from that seen in adults. The diagnosis is to be made by the examination for tuberculosis elsewhere, and by the examination of the throat by the direct method. Chronic hoarseness in a child is more commonly due to a simple laryngitis associated with hypertrophied tonsils and adenoids, than to tuberculosis, while a few cases are syphilitic.

TUBERCULOSIS OF THE MEDIASTINAL GLANDS.

From what has already been said on the modes of infection by tuberculosis, the great importance of the mediastinal or bronchial glands in this disease must be admitted. We know that in the glands about the bifurcation of the trachea and at the roots of the lungs, the initial lesions of tuberculosis are often present. Also we are able to trace the infective processes spreading from these glands into the lungs, and there is no doubt at all that many and probably most cases of general tuberculosis, originate from an infection of these glands. Further, we know that caseous deposits in them may become healed, inasmuch as calcification is not rarely found in the post-mortem examination of these structures.

It becomes, therefore, a matter of great importance to consider what signs and symptoms may give rise to a reasonable suspicion of tuberculosis of these glands, for with prompt treatment there is some hope that a cure may be effected.

The glands on the right side are usually more severely damaged than those on the left, and should there arise perforation of a bronchus, it is nearly always the right bronchus or one of its branches that is affected.

The Diagnosis of tuberculous bronchial glands can rarely be made with certainty, but the condition may be suspected from the following signs and symptoms.

In the first place, the child is out of health, is losing flesh and becoming pale, the appetite is going, and perhaps there is some rise of temperature at night. In conjunction with such indefinite symptoms as these, the child may have a tuberculous appearance, with long eyelashes, hair coming far down on the temples, and with a downy growth of hair between the scapulae; or may be one of a tuberculous family. Such a condition, especially in the absence of any chronic indigestion or constipation, may lead one to suspect tuberculous bronchial glands. On examination, the veins at the side of the manubrium may be enlarged, a sign of importance only if unilateral. On percussion there may be found relative dullness over the manubrium and at the sternal ends of the intercostal space here. Dullness posteriorly between the scapulae is a sign of later development and greater significance. Of more value, but only found in well-marked cases, is definite increase of resonance felt in percussing or palpating the manubrium. The "Ewart-Smith bruit" is, according to some, a sign

of importance, although it is certainly not pathognomonic of enlargement of the mediastinal glands. It is heard below the sternal ends of the clavicles when the child's head is fully extended. It is described as being due to pressure upon the left innominate vein by the glands, which become tilted forward with the head in this position. Signs of pressure upon a bronchus, shown by poorness of air-entry in one lung or part of a lung, may be present. Should perforation of a bronchus have occurred, signs of consolidation of a part or the whole of one lung may be found. In the latter case the entire lung is rasping and, by causing very resistant distention on percussion, may closely simulate a pleural effusion. As has been stated, the right bronchus is much more commonly perforated by enlargement of the mediastinal glands than is the left. In some cases of tuberculous bronchial glands, a cough very like that of pertussis develops. It differs, however, from the true cough of that disease in that no whooping inspiration is present. A series of expiratory coughs is given: but just as the crowing inspiration is expected, the cough suddenly ceases. Sometimes puffiness of the face, occurring first round the eyes, is seen in cases of enlarged bronchial glands. Very rarely the glands may be felt by the finger pressed down behind the mandibulum.

The results of tuberculous of the bronchial glands have for the most part been mentioned. Very commonly, general tuberculosis originates from a focus in these glands, while the lungs may become infected by processes spreading out from the glands or by the perforation of a bronchus. Occasionally, the contents of a tuberculous gland getting into a bronchus become lodged in the glottis, and cause death from asphyxia, an accident which the author has seen once. Perforation of the œsophagus may occur. Dr. Still has recorded cases in which the softened glands pointed externally through or near the mandibulum.

The Prognosis must necessarily be very guarded. We have ample proof that recovery may take place; but we cannot possibly foresee the onset of any of the dangerous results which may arise. We can only have a suspicion by the general result of treatment, as to whether the glands are tending to soften or to heal.

Treatment should be adopted at once in any case where there is a reasonable suspicion of tuberculosis of the mediastinal glands. It must be undertaken on the general lines already indicated (p. 100).

PULMONARY TUBERCULOSIS.

In dealing with the various forms of tuberculosis of the lungs in children, one has to note at the outset the great preponderance of acute processes over those of a more chronic nature as seen in adults. Thus, acute miliary tuberculosis and acute tuberculous pneumonic conditions are common in childhood, while the more chronic forms,

comparable to the "pneumonia" of adults, are most uncommon in children. In the same way that we find local preliminary signs of resistance to the infection are noticeably absent in children, so we have to note the great tendency to a fatal generalization of the infection, particularly in children under the age of five years. Tuberculous processes in the lungs in children then, as compared with those in adults, tend to be more acute and more diffuse, and to give rise to a general systemic infection.

Another difference is seen in the way in which the lungs become involved. In children, the initial pulmonary lesions are rarely found localized at a spot just below the apex of the lung. More commonly the infection starts from the glands at the roots of the lungs and spreads into the tissues of the lung, affecting a fan-shaped area irrespective of the divisions of the lungs.

Again, infection of a lung or part of a lung through perforation of a bronchus by a caseous gland is of common occurrence in children. Dr. Still found it 25 times in a series of 200 autopsies on tuberculous children; as 21 of these the right bronchus was affected. This probably explains the fact that infection starting at the base of the lung is not so uncommon in children as in adults.

Cavitation in pulmonary tuberculosis in children is by no means the rare condition that is often supposed, but such a process is nearly always an acute one, with little of the fibrotic changes that are usual in the case of adults.

As a general rule, it may be stated that any case in which well-marked pulmonary signs have existed for more than a year without causing death, is not of tuberculous origin.

Clinical Varieties.—The following main groups of cases may be recognized:—

1. **Acute Miliary Tuberculosis.**—A certain amount of diffuse miliary tuberculosis is present in the lungs, as in the other organs, in cases of general tuberculosis. Unless the lungs are thickly studded with tubercles, no clinical evidence of pulmonary disease is obtained. When of sufficient severity, there are seen to be dyspnoea, great pallor, and some cyanosis, while there are usually sustained fever and considerable enlargement of the spleen. The actual signs in the lungs are, however, not distinguishable from those of acute bronchitis. There is no sputum, unless older pulmonary lesions are present.

The diagnosis has to be made by the symptoms of the disease and enlargement of the spleen, rather than by the pulmonary signs. In infants, acute miliary tuberculosis of the lungs is always associated with tuberculous meningitis; in older children this, as a rule, very soon develops and causes death.

2. **Acute Tuberculous Broncho-pneumonia.**—This type closely simulates simple broncho-pneumonia. The onset may be rapid, and

accompanied by high fever and much prostration, while the physical signs in the lungs are those of areas of consolidation. The diagnosis of the tuberculous origin of the disease can hardly be made at first, but as the expected improvement does not occur, the true nature of the infection may be suspected. Even then it is to be remembered that many non-tuberculous pneumonias do not clear up in the usual time, and that there are many possibilities to consider (p. 57) before the case is confirmed as tuberculous. Definite signs or symptoms of tuberculous meningitis will render the diagnosis clear. Any considerable enlargement of the spleen is in favour of a tuberculous pneumonia; but it is to be borne in mind that in severe and protracted pneumococcal cases this organ is often increased in size.

In some of the cases in this group a more prolonged course is run. The areas of consolidation increase in size, and softening and cavitation may develop. The patient may appear to have a succession of attacks of broncho-pneumonia, improving in the intervals of fever, or there may be a steady downward progress from the beginning. The length of the illness may be from a fortnight, in very acute cases, to several months in the more chronic instances.

5. **Early Cases.**—As has been mentioned, in children the infection often spreads from the mediastinal glands into the tissue of the lungs. In such cases the physical signs are to a large extent only those of enlarged mediastinal glands (given on p. 129), to which perhaps may be added increase of the dullness at the sides of the mammae, and particularly, dullness in the interscapular region posteriorly, together with scattered crepitations in the neighbouring parts of the lung.

In this form, if the patient comes under treatment at an early date, recovery is certainly possible. In giving a prognosis it must be remembered (in this group of cases particularly, although the rule holds good for all cases of pulmonary tuberculosis in children) that the actual amount of pulmonary infection is likely to be considerably greater than the physical signs would suggest.

4. **Infection by Perforation of a Bronchus.**—This is of such comparatively common occurrence, that where consolidation appears sharply confined to one large portion of a lung, such a condition should be suspected. Very often in these cases such resistant dullness and such diminution of breath-sounds are present over the diseased portion of the lung that pleural effusion is diagnosed. The mistake is especially apt to be made where a main bronchus has been perforated and an entire lung flooded with caseous material. As has been mentioned, the right bronchus is very much more commonly the seat of perforation than the left.

Sometimes the patient lives long enough for softening and cavitation to occur; but usually death takes place within a few weeks from a spread of the disease in the lungs or to the meninges.

5. **Fibro-caseous Tuberculosis.**—This form of infection, which is the extreme form of adults, is very uncommon in children. It occurs

only in children of over five years of age, and is rare in those under ten years, when tuberculosis is not at all frequent, and it forms only a proportion of the cases of pulmonary tuberculosis in them. The physical signs are similar to those of "phtisis" in adults. The sputum contains tubercle bacilli. Hemoptysis may occur, but is uncommon.

As in adults, a fair proportion of cases coming very early under treatment become arrested; but as a whole, these cases do not do so well as those of a later age, and death usually occurs within a year of the discovery of the disease.

6 **Chronic Tuberculous Interstitial Pneumonia.**—Any condition in which there is much fibrosis and contraction of the lung is very rarely of tuberculous origin in a child, and such a diagnosis should not be made unless tubercle bacilli are present in the sputum. Such conditions are usually post-pneumonic in origin.

Diagnosis.—This has been already discussed to some extent, and only a few further points need consideration.

Hemoptysis in cases of pulmonary tuberculosis is of rare occurrence in children. Under the age of six it is extremely rare, but it is occasionally seen in cases of general tuberculosis in which there are added septic changes in the lungs. In older children it arises in connection with the pulmonary tuberculosis of the adult type. Even here, however, it is uncommon.

Night-sweats are of very little diagnostic value in children, as so many children who are ill in other ways suffer from them.

Vomiting may be due to the violence of the cough, to gastric set up by swallowed sputum (practically never to tuberculous ulceration of the stomach), to peritoneal adhesions, or to the onset of meningitis.

In many cases of intestinal indigestion of the type called "mucous disease," a mistaken diagnosis of pulmonary tuberculosis is made. This is referred to elsewhere.

Often it happens that the lungs, after an attack of bronchitis or pneumonia, do not recover completely by a cure. A few sharp crepitations and patchy areas of blowing breathing and impaired resonance remain, and a diagnosis of tuberculosis is made. In a few weeks the child gets perfectly well, and the exact nature of the case can never be known for certain. Such instances teach us to give the child the benefit of the doubt, and not to fall back upon the diagnosis of tuberculosis in undue haste. Similarly, in the acute cases it is to be remembered that all cases of pneumonia in which the temperature remains somewhat raised are not necessarily tuberculous (p. 93).

Again, in cases of chronic pulmonary disease, such as chronic bronchitis and bronchiectasis, an erroneous diagnosis of tuberculosis is often made. Such mistakes should be avoided by remembering the rarity of such forms of pulmonary tuberculosis in children, by the examination of the sputum, and by watching the course of the

disease. Where widespread primary signs have existed for a year without causing death, tuberculosis can almost certainly be excluded as the cause of the disease.

The sputum in children is usually swallowed, and so not available for examination. Dr. Holt, however, has met with success in obtaining sputum from even quite small children by exciting a cough by tickling the pharynx with a piece of muslin wrapped round a pair of artery forceps. By this means sputum is coughed up and collected on the muslin, and can be examined.

The value of Calmette's *ophtalmic* or Von Pirquet's cutaneous tests is considerably reduced by the fact that they may be positive, owing to some latent glandular tuberculosis so common in children, which has nothing to do with the illness from which the child is suffering. Of the two tests, the latter is the better, as the *ophtalmic* reaction occasionally produces a very severe conjunctivitis or even corneal ulceration. Mucous children do not react to these tests, nor do all cases of acute miliary infection. In doubtful pulmonary cases, excepting acute miliary cases, observations of the opsonic index before and after breathing exercises are of much greater value. Should such exercises cause fluctuation in a previously constant index, it is highly probable that there is an active focus of infection in connection with the lungs. In pure acute miliary cases the opsonic index usually keeps fairly constantly about normal.

Treatment.—The greater part of the treatment for pulmonary cases has been already described under the general treatment of tuberculosis (p. 110).

Cod liver oil may be given in doses of 1 to 4 minims in an emulsion of cod liver oil. It is necessary that it should not be allowed to damage the digestion and cause loss of appetite. It may also be used in the form of an inhalation, which is particularly useful in the fibrinous form of older children, especially where there is any secondary infection of cavities. In these cases it soothes the cough and reduces the fever, and its exhibition is often associated with an improvement in the appetite and an increase in weight. It may be used on a Barney-Yeo's inhaler, which is worn continuously day and night, except at meals. Dr. Lee gives the following formula, 6 drops of which should be put into the inhaler every hour: 2 drachms each of carbolic acid, creosote and spirits of chloroform, with 1 drachm each of tincture of iodine and spirits of ether. Children take very cheerfully to this treatment, and soon cease to object to their "muzzle," as they term it.

TUBERCULOUS PLEURISY.

Dry Tuberculous Pleurisy is but a prominent disease in childhood. It shows no difference from the same condition in adults, and as a rule, after a day or two, passes on into the stage of effusion.

Tuberculous Pleural Effusion.—Although in pleural effusions the serous are greatly outnumbered by the parient, nevertheless it is not uncommon to meet with cases of the former in older children. A serous pleural effusion in a child is nearly always of tuberculous origin.

The onset is as a rule rapid. There is pain in the side for a short while, associated with fever, and very quickly effusion develops, causing dyspnea and cyanosis. With two or three days' illness the chest may be found full of fluid. Less often, the condition arises insidiously, the patient becoming pale and wasted, but not seeming sufficiently ill to be put to bed.

The physical signs and diagnosis of pleural effusion in children are given under the more important disease of empyema (p. 102).

As to the differentiation between serous and parient effusions, there is little that can be said. It is generally unsafe to hazard an opinion



FIG. 27.—TUBERCULOUS PLEURISY: moderate tuberculous pleural effusion. (Note, the glandular swelling was emphasized by shading for photographic purposes.)

on the matter. Where a child has been watched through an attack of pneumonia and the pleural effusion develops under observation, it is safe to diagnose it as parient. But when, as so often happens, the child is seen for the first time after a week's illness, and is then found to have an effusion, it is almost impossible to go by the history of the case: for as has been said, a tuberculous effusion may originate suddenly with high fever, dyspnea, cough and pain in the chest, so that what seems to be an empyema following pneumonia, may easily turn out to be serous effusion. The localized bulging of a pointing empyema is of course definite evidence of pus, but a diffuse swelling of the affected side may occur in tuberculous cases. A sign which Dr. Still has pointed out to the author is that of enlargement of an intercostal gland on the affected side (Fig. 27). This is more common in serous than in parient cases, but as it is often absent in the former, its absence is not an indication of an empyema. Such an

enlarged gland is often better felt than seen. The examination of the blood gives the best guide; but even this is not infallible. A high leucocytosis, especially when the polymorphonuclear cells are proportionately increased, is very strongly in favour of the effusion being purulent. Rapid disappearance of the fluid is in favour of a serous effusion.

As a rule then, even if the diagnosis of fluid in the pleural cavity is fairly made, it is wisest to explore the chest to ascertain the nature of the effusion. The cells present in the fluid should be examined. In tuberculous cases the majority of them are lymphocytes.

Prognosis.—In a child the prognosis is not so good in the case of a tuberculous pleural effusion as in an adult. Although the acute symptoms as a rule pass away quickly, the ultimate outlook is not good owing to the great dangers of recrudescence of the infection in the lungs or elsewhere.

Treatment.—This does not differ from that used for the disease in adults. As it is usually wisest to explore the chest with a needle to make certain of the character of the effusion present, so it is best, if clear fluid be found, to proceed to aspirate the chest. Many of the slighter cases, however, clear up without aspiration by rest in bed and the administration of saline aperients. If the fluid repeatedly recurs, an operation as for empyema may be performed.

As soon as possible the child should be put under the best possible conditions for getting entirely cured (p. 119).

TUBERCULOSIS OF THE CIRCULATORY SYSTEM.

Only two conditions need consideration here, namely, tuberculous pericarditis and endocarditis. Of these the former is much the more important.

TUBERCULOUS PERICARDITIS.

It is not very uncommon to find solitary tubercles upon the heart in autopsies on children who have died of general military tuberculosis. In such a connection the disease is of no clinical interest.

On rare occasions, tuberculous pericarditis is clinically recognizable, and is then a matter of considerable interest. It is usually seen in older children. A soft friction is audible in some cases, but the disease is remarkable amongst diseases of the heart for the amount of pericardial effusion which may be caused by it, and it is to be noted that this is not associated with much dilatation of the heart. It differs then at once from rheumatism, in that myocarditis here is not a prominent characteristic of the disease. Similarly, endocarditis is not as a rule present, and no murmurs are heard. The physical signs are those of pericardial effusion, and this is the only type of pericardial

effusion that can be diagnosed in children with fair ease. The deep cardiac dullness is much increased in size, the lateral and upper borders all being displaced. The heart-sounds are very distant, and may be quite inaudible. Percussion over the heart reveals a very resistant dullness. Combined with these signs it is notable that the pulse is not the characteristic very rapid and fluttering pulse, such as would be the case were the increase of the deep cardiac dullness due to, or associated with, marked dilatation of the heart.

The symptoms are those of fever, pallor, and dyspnoea, to which may be added some precordial distress. Puffiness of the eyes, such as is seen in rheumatic pericarditis, may be present to a marked degree.

The pericardial effusion is similar to a tuberculous pleural effusion; it is clear, contains an excess of lymphocytes, and although tubercle bacilli can rarely be demonstrated in it, it will produce tuberculosis when inoculated into a guinea-pig.

Tuberculous pericarditis may be associated with recognizable tuberculosis elsewhere, and appears particularly to be related to that form in which serous membranes are chiefly involved, which is known as multiple serositis or polyserositis.

Multiple serositis is a condition in which the peritoneum, pleura and pericardium are affected, and produce effusions. It may be due to tuberculosis, chronic adhesive mediastinitis, renal disease, or very rarely in children, to carcinoma of the liver. In the tuberculous form, the disease may develop first in the peritoneum, and abscess be produced; after which the pleural cavities are attacked and lastly the pericardium. But it would appear that in certain cases the pericardium is the first serous membrane affected, and it is probable that the instances of tuberculous pericardial effusion which are occasionally met with in which no other serous effusions are present, will become cases of multiple serositis of this type. The course of a case of tuberculous multiple serositis is very uncertain. It may be rapidly fatal, or may be compatible with an invalided life of a few years. Death is usually due to meningeal involvement.

Tuberculous pericarditis, then, is often associated with other tuberculous effusions, and in their absence careful examination of the abdomen should be made for such an early sign of tuberculous peritonitis as a matted roll of omentum.

The Diagnosis of this form of pericarditis with effusion is not a matter of great difficulty granted that the possibility of the condition be remembered and a careful routine examination of the chest made. The puffiness of the eyelids may suggest the possibility of pericarditis. The tuberculous form differs from the rheumatic in that the child looks tuberculous and not rheumatic; there is no history of prior fits in the limbs, while there are no nodules nor any signs of fulginess or latent chorea. The examination of the heart shows a maximum of pericardial effusion with a minimum of myocardial change, as has

been emphasized already. Signs of tuberculosis may be found elsewhere in the body.

Prognosis.—It is possible that this disease may become arrested. Occasionally, one finds at autopsies upon cases of general tuberculosis in children signs of tuberculous pericarditis of old standing. Of such cases, however, we know little. From what has been said of the possible relationship of the disease to multiple serositis, it would be wise to give a very guarded prognosis, even where effusion is only present in the pericardium. In the few cases I have seen, pleural effusion has been present from the first, or has developed sooner or later. One cannot, therefore, but regard the consequences as likely to be very serious.

Treatment.—The pericardium may require tapping. A pint or more of fluid may be withdrawn. Apart from this, general treatment (p. 116) should be adopted.

TUBERCULOUS ENDOCARDITIS.

This is of very little clinical importance. Tubercles are not very uncommonly found just in front of the aortic endocardium in cases of general tuberculosis. The valves are very rarely attacked. In cases of general tuberculosis, an apical systolic bruit is very occasionally found to be due to tubercles of the mitral valve.

TUBERCULOSIS OF THE DIGESTIVE SYSTEM.

Abdominal tuberculosis is perhaps the most convenient term to use in connection with tuberculosis of the digestive system, for we have three forms of tuberculosis to consider, which are usually all present at the same time; namely, tuberculous peritonitis, tuberculous of the mesenteric glands, and tuberculous enteritis. As it often happens that the one type of infection is more severe than the others, separate consideration must be given to each of the three groups of cases.

Abdominal tuberculosis is found in 85.3 per cent of autopsies upon cases of tuberculosis (Still). Such lesions in the abdomen as have been described as dealing with general tuberculosis are not however recognizable clinically, and thus abdominal tuberculosis very often exists without giving rise to any characteristic symptoms. Cases of abdominal tubercle are, however, common enough, and some of them can only be thus termed (until the infection becomes generalized), inasmuch as there is clinical evidence of severe peritoneal, glandular, and intestinal disease. Usually, however, cases can be correlated to one or other of these three groups according to the part chiefly affected. There is no harm in this if it be remembered that pathologically all three sets of lesions are usually present to a greater or less degree, and for descriptive purposes such a division is quite necessary.

TUBERCULOUS PERITONITIS.

In infancy, tuberculous peritonitis is said to be uncommon by some observers, but Dr. Stiff's statistics show that this is not so, and that death occurs from this condition as in other forms of tuberculosis, most commonly in the second year of life. The peritoneum may become infected from the mesenteric glands, but often it would appear from post-mortem evidence, that the affection has been from other sources, such as the lungs or bronchial glands. A history of injury to the abdomen is occasionally present, and is possible an exciting cause of the disease.

The clinical types of tuberculous peritonitis are two in number, the plastic and the ascitic, the former is the more frequent.

The early symptoms of tuberculous peritonitis are as a rule not very definite. There may be loss of appetite with occasional colicky pains in the abdomen, irregularity of bowels, constipation alternating with



FIG. 11.—TUBERCULOUS PERITONITIS, MORRIS CHILDREN. ABDOMEN AND UTERUS ENLARGED. ENLARGED SUPERFICIAL VEINS ARE WELL MARKED.

diarrhoea, and perhaps some slight rise of temperature. The characteristic symptom of the disease—namely, swelling of the abdomen—gradually becomes noticeable (Fig. 28).

In the plastic type, the abdomen is hard, and presents a peculiar doughy feel to the examining hand, a feeling of softness and inelasticity. Often a characteristic "omental mass" is present. This consists of a firm, sometimes nodular tumour running across the abdomen just above the umbilicus, and passing upwards towards the left hypochondrium. Owing to its direction and its firm consistence, it may be mistaken for the lower edge of the liver, but this can usually be made out with careful palpation at a higher level separate from the tumour. In its earliest stages the omental mass consists of an ill-defined soft tumour in the same position. The mass is due to thickening and calcation in the omentum.

Later, the child wastes considerably, and the abdomen becomes

further enlarged and much less soft to the touch. Through the thin abdominal wall, perpetual peristalsis may be seen going on in the coils of intestine, due to the partial obstruction from adhesions. Total obstruction may occur. The umbilicus becomes flattened, untended, and often indurated and reddened round its margins. At the umbilicus may develop a fistula through which caecocolic material or faeces may escape. The enlarged mesenteric glands may be palpable. Abscess may develop.

The ascitic form is characterized by the development of fluid and should this be much in amount, all other signs of tuberculosis in the abdomen may be masked. The veins running over the abdomen are enlarged (Fig. 28). The spleen may often be felt to be enlarged, and evidence of tuberculosis may be obtained in many cases by examination of the chest or cervical glands.

Diagnosis.—The plastic form must be differentiated from rickets and morbus coeliacus, in both of which the abdomen is enlarged.

The tuberculous abdomen is recognized by its peculiar doughy consistency in the early stages, and in the later by the taut state of the abdominal walls. Further, enlargement of the mesenteric glands or the presence of an omental tumour may be recognizable. The spleen may be enlarged in both tuberculosis and rickets, but considerable enlargement (two inches) is in favour of the former. A careful examination should be made for signs of tuberculosis in the lungs, cervical glands, or scrotum.

The ascitic type is recognized in the same way: it is to be remembered that tuberculous peritonitis is much the most common cause of ascites in children, syphilitic, arthritis of the liver being next in order of frequency. Signs of tuberculosis or of inherited syphilis elsewhere in the body should be carefully looked for.

Prognosis.—The outlook in the ascitic cases is generally held to be much better than in the plastic group. This is only partially correct. It is quite true that the ascites usually disappears, but it usually always leaves some plastic peritonitis which may or may not clear up.

In the plastic cases, the slighter instances probably get quite well, and the more severe ones improve very considerably under treatment on a rule. It is very difficult, however, to say that the condition is cured, and quite impossible to foresee whether further disease will show itself later, in the abdomen or elsewhere.

As in other forms of tuberculosis, the prognosis is worse in infants than in older children.

Sometimes great improvement occurs in what are seemingly hopeless cases. Even when a faecal fistula has developed, it may close and the child gain weight and put on flesh. The discharge of caecocolic material at the umbilicus may be of great benefit, and improvement commencing from that time.

TUBERCULOSIS OF THE MESENTERIC GLANDS.

In the majority of tuberculous children the mesenteric glands are infected, but in many such we cannot recognize any enlargement of them during life. Appear, the mesenteric glands may become palpable in cases of severe tuberculosis of the lungs or peritoneum. The group of cases, however, that we have to consider here, is comprised of those in which the mesenteric glands seem to bear the brunt of the infection, in which there is little or no evidence of pulmonary or peritoneal tuberculosis; but in which, from the disease in the glands, there are symptoms of ill-health. To this group the name "tuberc mesenterica" may be given; but unfortunately this term has been so loosely used that it has come to be applied to any form of abdominal tuberculosis, or even to simple non-tuberculous wasting in infancy.

This form of tuberculosis is not a common one. The symptoms are those of wasting, colicky pains, irregularity of the bowels, poorness of appetite, and slight fever, the same symptoms as those seen early in all forms of abdominal tuberculosis. On examination of the patient, enlargement of the mesenteric glands is found without peritoneum of appreciable extent. The tumour is usually felt either just to the left of the umbilicus, or in the right iliac fossa. It is often slightly tender on pressure.

The glands have to be distinguished from focal masses, chronic appendicitis, and ileocaecal tuberculosis.

Prognosis.—Some of the slighter cases probably get quite well, and a certain amount of improvement is often seen in the severer ones. Extensive alteration of the intestine makes the outlook very grave, and in cases in which there is very great enlargement of the glands this is likely to be present. Even if the infection becomes arrested there are dangers of recrudescence, generalization, and the formation of adhesions. Occasionally the caseous mass of glands empties itself through a fistula at the umbilicus, less often through an opening into the intestine.

TUBERCULOUS ENTERITIS.

This, again, is more common pathologically than clinically. In many cases it is associated with obvious tuberculous peritonitis and mesenteric adenitis, and where severe it renders the prognosis of the other forms of abdominal tuberculosis very serious. It saps the patient's strength by the diarrhoea it causes, and is a source of further infection to the glands and peritoneum. Further, it may cause perforation of the intestine, adhesions or short-circuitings between various coils of intestine.

Tuberculous enteritis may, however, exist in some degree of severity without there being any obvious peritonitis or even much enlargement of the mesenteric glands. Such cases may be very puzzling. The child suffers from alternate attacks of constipation and diarrhoea, the

latter tending to get gradually worse. The stools are watery and very offensive. There is some fever with abdominal pain and progressive wasting. The abdomen is examined, and nothing abnormal is found beyond perhaps a slightly enlarged and fatty liver. Sometimes, as the result of the diarrhoea, oedema of the extremities develops and further complicates matters. On watching the case, however, the diagnosis becomes clearer. The diarrhoea does not react well to treatment, the spleen becomes enlarged, the mesenteric glands increase in size, and perhaps spots of tuberculosis appear in the lungs. Such cases as these are, however, infrequent. As a rule tuberculous enteritis is only part of a recognizable abdominal tuberculosis.

The ulcers are most commonly found in the termination of the ileum and the cecum, but they may extend from the duodenum downwards into the colon.

A form of tuberculous enteritis which is of some interest is that sometimes known as ileocecal tuberculosis. There is a palpable tumour in the right iliac fossa. The disease simulates chronic appendicitis or partial obstruction of the gut, and is not infrequently found at operations for such conditions as these. Complete removal of the affected part may be practised. Usually it is wise to shunt circuit it first, giving it time to shrink in size before it is actually excised.

Treatment of Abdominal Tuberculosis.—In addition to the general principles given on p. 139, some special points require mention.

Rest in bed is the first requisite, for by this means excessive stimulation is avoided, and diarrhoea, if present, is to some extent lessened. In most cases of abdominal tuberculosis mercurial invasion of the abdominal wall is ordered, while some speak in favour of iodoform ointment. Cresate may be given internally, 4- to 5-minim doses being added to the cod-liver oil emulsion, or, if diarrhoea be present, to a carrier-oil mixture.

If no diarrhoea be present, cod-liver oil, cresate, and sulfate of iron are the most useful drugs to prescribe. The food should be as plentiful as the child can digest. Milk, eggs, cream, milk-puddings, minced meat, etc., may be given. Raw meat juice may also be of benefit.

In the presence of diarrhoea, intestinal detention, flatulence and colic, a different dietary, from which oily and fermentable foods are omitted, is required. Cod-liver oil must be withheld. Dr. Sutherland has devised the following régime which gives admirable results.

(a) While the appetite is feeble dilute soups made from mutton, beef, chicken or veal should be given. These may be strengthened by the addition of raw meat juice (one or two ounces daily), sarsaparilla or plasmon powder. During this stage castor oil should be administered in order to clear the bowel of all fermenting debris.

(b) As the appetite improves, irrespective of the other symptoms, the diet may be increased, but should be constituted on the same lines

as the foregoing: For breakfast, fresh fish, tongue, slice of egg fried or lightly broiled, with two small pieces of toast and a teaspoonful of weak cocoa containing an ounce of milk. For dinner and supper, fish, chicken, sweetbread, tripe, mutton, beef, boiled or roast, hot or cold, no fat, sauce or gravy; breakfast or plainest biscuits. Half a glass of claret may serve as a tonic and as a detergent to the diarrhea. Water may be freely taken between meals.

With such a diet as this the patient does not put on fat, but nevertheless gains in strength while the diarrhea and other abdominal symptoms tend rapidly to disappear.

(c). With the subsidence of the abdominal symptoms and the improvement of intestinal digestion, fat and nourishing foods may be slowly and carefully re-introduced into the diet. The fat may be supplied in the form of cod-liver oil emulsion, yolk of egg and cod-liver in oil.

For drugs, during the first stages a castor-oil mixture may be given until the intestine is cleared of food debris. Should the diarrhea persist, bismuth in large doses, with or without opium, is of considerable value. Chalk, catechu, hematoxylin, iodo, iron, silver nitrate and tannin may be tried for this symptom. In the checking of the diarrhea, however, the main reliance should be placed upon the protein diet.

Operative Treatment.—Taking first the question of operation in the ordinary cases of tuberculous peritonitis with ascites. Formerly it was a widespread custom to have these cases operated upon and the fluid removed; but to a large extent this has been given up. It is undoubted that in the majority of cases of this type of the disease, the fluid tends to disappear under medical treatment, and that there is no advantage to be gained by an operation. Indeed, in some statistics the influence of operative treatment seemed harmful. In tuberculous peritonitis, operation then should only be undertaken for the relief of some particular symptom, such as obstruction, excessive distension or abscess formation.

When a small group of caseous glands is causing pain and partial obstruction, it may often be successfully removed. Operation may be necessary for obstruction to the intestine by means of a band which has resulted from a slight previous attack of tuberculous peritonitis. Mention has already been made of the successful operative treatment in some cases of ileocecal tuberculosis.

TUBERCULOSIS OF THE GENITO-URINARY SYSTEM.

There is little of medical interest in this subject which is in any way peculiar to childhood.

RENAL TUBERCULOSIS.

The most common lesion of this nature is the occurrence of military

tubercles in cases of general tuberculosis. These are usually superficially placed, less often they are in the substance of the organ. Sometimes minute infarcts are produced by this process. The number of ciliary tubercles found in the kidneys is nearly always less than that in either the spleen or liver. Occasionally, in the same cases there may be a caseous mass of fair size in the kidney.

Tuberculosis of the kidney of clinical interest is of uncommon occurrence in children. The symptoms do not differ in any way from those seen in adults. The renal condition is practically never the result of an ascending urethritis from an infected bladder.

TUBERCULOUS CYSTITIS

This, usually the result of an infection from a tuberculous kidney, is of rare occurrence in children. It causes the same symptoms as in adults.

TUBERCULOUS EPIDIDYMITIS

It is worth while remembering that this is not a very uncommon disease even in quite young children, and may be of considerable diagnostic value in some doubtful cases of disease elsewhere in the body. It has to be distinguished from syphilitic orchitis.

III.—THE RHEUMATIC INFECTION.

Introductory.—Amongst children's diseases, there is none more worthy of close study than acute rheumatism. When we consider its frequency—rivaling with that of the tuberculous and pneumococcal infections—and its harassing and crippling effects, its importance becomes manifest, and the fact that it produces but seldom an immediately fatal result, must not hide from us the vast amount of damage it does amongst children. There is still much work to be done in connection with the rheumatic infection; much yet remains to be learned about its frequency, specificity, bacteriology, morbid anatomy, and true clinical manifestations. Perhaps no problem connected with the disease is more urgently requiring solution at the present time than that which has to do with its treatment by the salicylic acid preparations. To all these matters it is necessary to-day to make brief allusion in the description of the disease.

As seen in children and in adults, rheumatism shows many differences. The study of the disease has in the past been much retarded by considering it as an infection of adults showing peculiarities—the so-called "complications"—when it occurs in children. The true significance of the disease is appreciated when it is regarded as a children's disease, with cardiac, nervous, and other manifestations

which are less apt to be present in older subjects. In children the arthritis is of very minor importance. It is frequently absent, or may be replaced by aricular pains. Sweating is very little seen, the fever is usually slight in amount, while hyperpyrexia is extremely rare. On the other hand, cardiac rheumatism, chorea, nodules, and rashes, are very much more common in young than in old subjects. The rheumatic infection, in a word, tends to become generalized in children, as do all other bacterial diseases.

Etiology.—The *age-incidence* of the disease shows that it is very rare during the first two years of life. Infants are in fact practically immune to the disease. From this time onwards the infection begins to get more frequent, but does not become common until the fifth year. The greatest liability to a first attack of rheumatism appears to be shown by children between the ages of seven and ten. The circumstance shows that there is a very slight preponderance of girls affected. The *seasonal incidence* closely corresponds to that of enteric fever, including January, however, with the autumnal months in the dangerous period of the year. A *family predisposition* to the disease is very common. Chadwick found it in 70 per cent of a series of cases seen in private practice; and he pointed out that where there is a history of rheumatism on both sides of the family, the child is apt to have a severe and obstinate form of the disease. Tuberculosis is also commonly found in the family histories of rheumatic children, but rheumatism and tuberculosis are very seldom found active at the same time in the same subject; indeed, there is some evidence to show that the one active infection tends to protect against the other. Other infectious diseases may predispose towards rheumatism. This is notably the case with scarlet fever, to which rheumatism can often be traced. Less frequently, measles and diphtheria are followed by rheumatism.

Certain localities, and probably certain homes, appear to be associated with a high incidence of rheumatism; but although it is not very rare to find more than one of a family attacked within a short time, the disease can hardly be looked upon as infectious in the ordinary sense of that term.

Bacteriology.—We must here take for granted that rheumatism is a bacterial disease, a point admitted on practically all sides, and the next consideration is as to whether or no it may be regarded as a specific infection. There is by no means unanimity on this question except amongst those who have studied the disease as it appears in children. Finding that rheumatism, at one after one in children, shows the same symptoms, tendencies and course, one becomes convinced of the fact that although allied in some points to scarlatina, erythema nodosum, malignant endocarditis, and rheumatic arthritis yet rheumatism remains in itself a disease which, if of bacterial origin, is

almost certainly due to one specific organism. Pathological evidence, both macroscopic and microscopic, supports this view. Inasmuch as rheumatism is associated with severe damage to the tonsils, it might be expected that secondary infections would not be rare.

A micrococcus, originally found in rheumatic subjects by Teichgraber and Wassermann, has been studied by Drs. Payton and Price, and has by them been termed the *M. rheumaticus* or *rheumococcus*. This they regard as the cause of the disease. The evidence for their view is very strong, and has been accepted by many clinicians; but as yet bacteriologists have for the most part refused to allow that this organism has been shown to be the specific causal agent.

The position of affairs in the controversy may be briefly discussed. The organism is a very minute, Gram-positive coccus, growing in pairs or short chains. During life, it has been found in the blood and articular fluid of patients suffering from acute rheumatism. Post mortem it has been grown from the blood, the joint fluids and tissues, nodules, tonsils, pericardial fluid, and endocarditis growths in some of fatal rheumatism. In chorea, it has been found during life in the cerebrospinal fluid, and has been seen after death in the perivascular spaces of the cerebral vessels. Experimentally, it has produced polyarthritis and pericarditis of a non-purulent febrile type, endocarditis, and rarely, nodules and choreiform movements. From the experimentally produced lesions it has been recovered, and has reproduced the same conditions.

On the other hand, there are two main objections urged against accepting this coccus as the causative organism of rheumatism. Firstly, it has been repeatedly rejected as absent in rheumatic lesions. The would, however, only tend to show faulty technique, or that rheumatism is not a bacterial disease, or that the organism quickly disappears. Secondly, it cannot be distinguished by laboratory tests from some of the streptococci inhabiting the alimentary canal, notably the *S. salivarius* and *faecalis*. Nothing, however, is more likely on clinical grounds than that rheumatism is due to an organism which is normally present in the digestive tract. It must also be added that the various sugar reactions which have been so largely relied on for the differentiation of the streptococci, have now been shown to be quite inadequate for this purpose.

Symptomatology.—The most important symptoms of rheumatism in children are sore throat, muscular pains, arthritis, and chorea, together with cardiac rheumatism.

A Sore Throat is so commonly associated with rheumatism that it is thought highly probable the infection enters through the tonsils, and that the tonsillitis is due wholly or in part to the rheumatic organism. The tonsillar affection may precede the rheumatic pains by very varying periods. Commonly, however, the rheumatism appears within a week or two, and often before the throat symptoms

have disappeared. The most frequent form of sore throat shows the tonsils swollen and with the soft palate reddened and presenting a glazed appearance, as though smeared over with the white of egg. Rather less common is an ordinary follicular tonsillitis, showing small patches of a creamy white exudate which occasionally run together, simulating a diphtheritic condition. It is the rule to find post-inflamed enlarged tonsils, in the depths of which are loci of semi-solid purulent exudate.

Muscular Pains frequently replace the typical arthritis of rheumatism as seen in adults. Any of the muscles may be affected. It is probable that the fibrous tissues are the seat of the inflammation rather than the muscles themselves. Most commonly these pains occur in the legs. They may be of a sharp character, coming on during exercise and causing the child to cry out and stop to rub his limbs, sometimes to stumble or fall. Such pains may only last for a few minutes, but are apt to recur. They are often referred to the back of the knee-joints, to a spot just in the middle of the popliteal space. Other rheumatic pains are more dull and aching in nature, and give rise to a sensation of stiffness in the affected part. When severe the child is unable to get about, and will often remain crying for long periods. Such pains as have been described are often called "growing-pains." They are, however, associated with a rise of temperature, and it must be remembered are truly manifestations of rheumatism, so that the heart is in danger.

Pains in the neck causing a rheumatic torticollis are very common. Pains in the muscles of the back may give rise to a suspicion of tuberculous caries of the spine. Pains in the arms are usually felt in the neighbourhood of the joints, and as a rule are quickly recognised as rheumatic. In the region of the hip-joint such muscular rheumatic pains may be very persistent, and rather differ in this way from the more fleeting pains round other joints.

Pains in the Sides and Epigastrium are very common in rheumatic children, and are often of sufficient severity to cause the child's visit to the doctor. The left side is rather more frequently affected than the right, and in some cases the pain is said to be worse after food. Thus it is possible that some of these pains are of a dyspeptic nature, but certainly the majority do not seem capable of this explanation, and may more properly be included in the group of muscular rheumatic pains arising from the intercostal muscles and the upper part of the rectus abdominis muscle. From their localisation they are often described as due to a "stitch." They usually disappear quickly when treated by a mixture of the salicylate and bicarbonate of soda.

Arthritis is less common, less obvious, and less painful in children than in adults. The large joints are those usually affected, but the proximal interphalangeal joints are very commonly attacked in children. The synovial membrane is often inflamed, but for the most part it is the peri-articular structures which are attacked.

Subcutaneous Nodules, rheumatic nodules as they are called, are of much interest and importance. They are most commonly seen round the patella, on the occipital ridges, and elbows; but they may be found over any of the bony points of the body: the knuckles, spine, scapula, etc., or along the course of the superficial tendons of the wrists and ankles (Fig. 19). Where very numerous they may be felt over the muscles of the back and abdomen. They also occur in the pericardium. Where few in

number they show a remarkable symmetry in their distribution.

In their most usual form they are small, painless, freely movable lumps, which are often better felt than seen. They arise rapidly and may disappear in a few days. They consist of small collections of fibrinous exudate, together with the formation of fibrillants which in the later stages are present in large numbers.

These nodules may also be seen in two other forms. Where they remain unabsorbed they become hard, and to some extent fibroid, so that they take long in disappearing. Their third form is that which may be called the haemorrhagic form of nodule. These are usually larger than the ordinary type, and appear over the bony prominences of the back. They arise very rapidly and are soft in consistence. The skin covering them is discoloured and tensed in appearance. They look like rapidly-growing sarcomata, but are distinguished by their position and symmetrical distribution, and by the associated or preceding rheumatism, which is usually of a severe type.

The clinical significance of rheumatic nodules in children is fourfold. Firstly, they are in their typical form quite distinctive of acute rheumatism. Secondly, their presence indicates the severe forms of cardiac rheumatism, almost invariably ends in pericarditis. Thirdly, generally speaking, in number they correspond to the severity of the infection, and where most copious, pericarditis is usually present. Lastly, they give a clue to what is happening in the valves of the heart: where they are rapidly absorbed, we may conclude that the valvular vegetations are also disappearing; where they remain and become fibroid, we may suspect similar sclerotic changes in the



FIG. 19. RHEUMATIC NODULES (ACUTE) OVER ELBOWS AND OCCIPITAL RIDGES. (LEFT AND LOWER ELBOW FIG. 19.)

valves. This mental association of the changes in the nodules and in the valves will be seen to be quite legitimate if we consider what position the nodules occupy in the disease.

Pathologically, rheumatic nodules take the same position in rheumatism that pyæmic abscesses occupy in pyogenic infections. The semi-gelatinous filamentous exudate which is found in the nodules is also found in the valvular vegetations in the pericardium, on the pleura and in the joints and peri-articular tissues. As Dr. Poynton has said, it is to be regarded as "rheumatic pus." It is, that is to say, the result of the reaction of the tissues to the rheumatic organism, and one which we cannot help regarding as a specific reaction to a specific organism. The type of nodule described above as the hemorrhagic, would seem to fall into line with the hemorrhagic forms of arthritis, pericardium, and pleurisy which are found in severe rheumatic infections. Taking these points into consideration, it is justifiable to conclude that the behaviour of the nodules may guide us in forming an opinion as to what is going on in those rheumatic lesions in the heart which we cannot directly observe.

Anæmia of the chlorotic type is very common in rheumatic conditions. As a rule, it is not severe, and improves quickly when the infection comes to an end. In long-lasting rheumatism, however, the anæmia may become much more profound, and in some instances is the most striking clinical feature of the case. The pallor of the patient gives an important indication as to the progress of convalescence, for where the infection is still present, although giving rise to little or no rheumatic pain, the anæmia is prone to remain unimproved by the ordinary hæmatinic drugs. There is, however, no truth in the saying that iron brings back the rheumatism; it is the omission of the salicylate rather than the administration of the iron, which is responsible for the recrudescence.

With the onset of acute rheumatism there is a moderate increase of the leucocytes in the blood (15-18,000 per c.mm.).

Nervous Symptoms have been recognised as associated with rheumatism in children for many years. Where they are very well marked, they form the condition known as chorea, which is described later; where less obvious, they may escape recognition. During both the acute and the convalescent stages of rheumatism, very definite nervous instability is commonly present. To such, the term latent chorea may be given, and the signs and significance of this are discussed under CHOREA (p. 167) to which the reader is here referred.

Headache is not uncommonly present during the prodromal stage of acute rheumatism. It is more constant in chorea than in muscular or arthritic rheumatism. In children it is nearly always due to the infection, and very seldom to the treatment by salicylate of soda. It is not necessarily dependent upon the anæmia.

Hyperpyrexia, with its severe cerebral symptoms, does not differ from the same condition in adults, save that it is still more uncommon.

The Temperature in acute rheumatism in children is as a rule lower than in adults, and seldom goes above 102° . Usually it falls to normal under treatment in the course of two or three days. In severe cardiac rheumatism, as in pericarditis, the temperature tends to be higher and to take a much longer time to reach normal. As the temperature begins to break, it becomes swinging in type, with a difference of a couple of degrees between the rising and evening readings. This passes from the remittent to the intermittent form, and gradually settles down to normal (Fig. 30).

The temperature of convalescence, so-called, is worth some study. It is intermittent in character, ranging from about 98° in the morning to 99° or 100° at night. This nocturnal rise of temperature may continue for many weeks, and is usually associated with a very slow

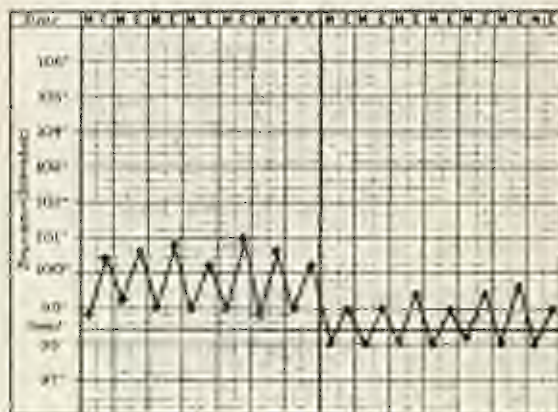


Fig. 30.—PATTERN OF TEMPERATURE CHART FROM A CASE OF CARDIAC RHEUMATISM, CHILD 5 YEARS.

The intervals from 98° to 99° (marked) point into the temperature level of convalescence (convalescent infection).

improvement in the general condition. It is very liable to be interrupted by periods of recrudescent or the infection, in which the temperature becomes further raised. In such relapses there may be the renewal of arthritic signs, or the development of tonsillitis or pericarditis, but in many there are merely increased pallor, further enlargement of the heart, and a rise in pulse-rate, giving evidence of fresh aggravaation. Such febrile relapses (Fig. 31) may be due to the child being allowed to get up (as in tuberculosis), while in other instances exercise has a steadying effect upon the temperature (again as in tuberculosis infection). Unfortunately, there is a general tendency to regard this intermittent temperature of convalescent rheumatism as harmless, unavoidable and normal; but it is certain that

the children who show it do not do so well as those in whom the temperature is more strictly normal. There is no reason why we should disregard this sign of bacterial activity in rheumatism any more than in tuberculosis.

We must conclude, therefore, that a regular swinging, intermittent temperature during the convalescent stage of rheumatism indicates a still smoldering infection, a bacterial activity which is damped down but not destroyed. Where it is present, it is therefore our duty to endeavour to counteract it by the further use of salicylate. It is generally possible by physical examination to locate the site of the active rheumatism: usually this is in the myocardium, but where there are no signs pointing to the involvement of any particular focus, it is not improbable that the infective agent is in the spleen.

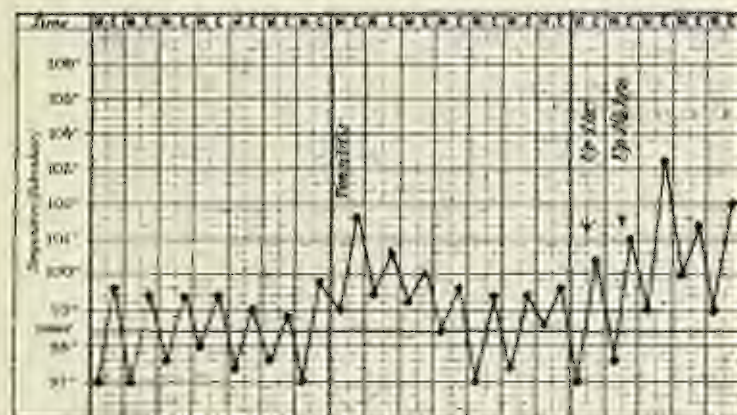


FIG. 11.—TEMPERATURE CHART FROM A CASE OF RHEUMATIC CARDITIS, MYOCARDITIS AND ENDOKARDITIS, AGED 12 YEARS.

The temperature shown in this chart is interrupted (1) by the usual remissions, and (2) by the effect of exercise.

Cardiac Rheumatism.—It is unfortunately necessary that the description of rheumatic heart-disease must be given divorced from the description of the acute rheumatic infection. It is dealt with in Section VII. It must suffice to mention here three points of fundamental importance in connection with cardiac rheumatism.

Firstly, some dilatation of the heart is practically invariably present at some time during the active stage of the rheumatic infection. This does not, however, necessarily imply the existence of any cardiac lesion. Secondly, usually speaking, it is the condition of the myocardium which is of greatest importance in rheumatic heart-disease in children, even where the pericardium and endocardium are involved. Thirdly, a cardiac breakdown, even in the case of long-standing

heart-disease is in a child almost invariably due to fresh cardiac infection.

Skin Manifestations.—The acid scaræ, so characteristic of the disease in adults, are very rare in children. Sweating of the face is occasionally noticeable. As a rule, the only sweating found in rheumatic children is that of the palms and soles, which are usually very moist, and may remain so for a long time.

A rheumatic erythema, generally conforming to the type of erythema marginatum, is well-known. It may closely simulate the rash of scarlatina.

It is probable that erythema nodosum rheumatica, or erythema nodosum, is essentially a rheumatic manifestation, although exceptionally they occur in rheumatic children. In such cases there is probably a dual infection. The hemorrhage occurring in connection with rheumatic nodules has already been described.

Rheumatic Pitting certainly exists pathologically, and as such is always associated with pericarditis. The pitting, usually at the base of the left lung, is covered with a fibinous exudate, sometimes hemorrhagic. During life it may give rise to a pleural transudate effusion.

Rheumatic Pneumonia is usually described clinically as being found at the base of the left lung and associated with pericarditis. It is probable that the physical signs in most of these cases are accounted for by compression of the lower lobe of the left lung by an enlarged heart. Even the finding here post mortem of small areas of broncho-pneumonia is no proof of their rheumatic origin.

That primary puerile or pneumonic conditions are ever rheumatic in origin is as yet unproven.

Albuminuria is frequent during the acute stage of rheumatism. It is usually quite transient. *Hæmaturia* under similar circumstances is very uncommon, but when it occurs seems to leave no permanent renal disease.

Bursitis, acute in character and resembling a septic condition, is a rare manifestation of rheumatism. I have seen it once in a case of acute rheumatism, the sub-deltoid bursa being painful and greatly swollen. Dr. Sutherland has kindly referred me to a case under his care in which an olecranon bursa became inflamed during an attack of rheumatic fever. In both instances, the swelling quickly disappeared under the influence of salicylate.

Iritis does not appear to occur in the rheumatism of childhood; a point of some theoretical interest.

Diagnosis.—The arthritis in children rarely causes any difficulty in diagnosis. Since rheumatism for all practical purposes does not occur during the first two years of life, the swellings of the limbs due to suppurative erysipelas and septicæ are easily distinguished from it. A suppurative condition may simulate a rheumatic joint somewhat closely; but the personal appearance of the child, the high

fever and leucocytosis, usually distinguish this condition even though the local signs be at first similar. A pseudo-paralysis due to the pain of rheumatism may be mistaken for acute poliomyelitis.

The muscular pains in the legs have to be distinguished from the sensation of the feet being "too heavy to lift," which is so often complained of by patients with dilatation of the left side of the heart. The two symptoms often co-exist in rheumatic children.

The most frequent mistake made in the matter of the diagnosis of rheumatism is that the true nature of the condition is overlooked. There is no little danger of the rheumatic origin of toxicities, febrile points, urticaria, headache and various nervous conditions being missed, and the symptoms being incorrectly interpreted.

Course and Prognosis.—The time occupied by an attack of acute rheumatism is very varying. The arthritis and muscular pains quickly yield to treatment, and in two or three days, or less, the patient is free of pain. The long cases are those in which the heart is badly attacked, and then the temperature may remain raised and the patient bedridden, for as long as three months. The occurrence of pericarditis, nodules, and profound anæmia, are indications as a rule that the infection is of a severe type and likely to prove intractable.

The prognosis is worse in children than in adults, for two reasons: the heart is very much more often damaged in young subjects than in older ones, and it is of course upon the condition of the heart that the prognosis both immediate and ultimate depends; secondly, in children there is the great danger of frequently renewed attacks. This great liability to fresh rheumatism, whereas to a large extent we attempt to assestate prognosis in heart-disease of children.

It must not be forgotten that it is not extremely rare for a first attack of rheumatism to prove fatal in a child.

Treatment.—Rest in bed is the first essential. By this means the number of heart-beats is lessened, the heart is rested, and its liability to permanent damage is diminished. The child should be kept warm between blankets. Local treatment for the arthritis, beyond possibly the strapping of the joints in need, is very seldom required. The external application of preparations of salicylic acid is of comparatively little use, as they are so slowly absorbed; whereas the drug given internally is very quickly absorbed, appearing in the urine within an hour or less.

The throat should be sprayed with an antiseptic preparation night and morning.

We come now to the vexed question of the value of salicylate of soda in the various manifestations of rheumatism; and at the outset it may be well to emphasize the fact that even if the drug has a specific action against the disease, it could not repeat the method

already done. Thus, it should not be expected to cut short the movements in cases of chorea running its definite course; nor would it follow that no deaths from heart-disease should occur during an acute attack of rheumatism; nor, one is tempted to add, would the drug of necessity take away cardiac murmurs.

It is generally accepted that salicylates relieve the pain and the swelling of the joints, and reduce the temperature of rheumatism in a way which is special to this disease; the point to be settled is the meaning of pyrexia in rheumatism.

In this matter we may, I think, hold the following propositions to be correct. First, that the temperature-chart is just as true a criterion of the activity of the rheumatic infection as it is of bacterial activity in other diseases. Secondly, that the reduction of the temperature in cases of rheumatic arthritis by means of salicylate is due, not merely to an antipyretic effect of the drug, but to a definitely antirheumatic or bactericidal property that this measure possesses. Thirdly, that it follows that if by salicylate we can lower the temperature in rheumatic manifestations other than arthritis, we are in all probability tending to destroy the bacterial activity of the infection.

As the view that salicylate has no beneficial action upon cardiac rheumatism is so widely held, we must consider it here for a moment. It must be remembered that the condition of the heart depends not only upon the bacterial activity present at the moment, but also upon the damaged state of the organ due to inflammation and toxic changes. Suppose that we could by salicylate put an end immediately to all active infection, still the heart would not necessarily there and then improve, for the damaged tissues require time to recover. Hence we could not expect clinical examination of the heart to give us definite evidence of immediate improvement. What then must be our guide? Again it is the temperature chart. When the temperature is normal, we must assume that the heart is in the most favourable condition to recover, and clinical experience proves that this assumption is correct.

The view that salicylate has no beneficial action upon active cardiac rheumatism must be given up. Already there are signs of its abandonment by many authorities. For years it was held that the drug must be dropped directly pericarditis developed, while now it is generally taught that in this condition salicylate is of service. To hold that a drug which has a beneficial action upon pericardial rheumatism has no influence upon the acute infection of the myocardium and endocardium, involves, surely, an intellectual strain too great to be supported for long.

In all forms of active rheumatism at the start we can lower the temperature by means of salicylate, but larger doses than those generally given are often needed.

In all forms of active rheumatism, therefore, we must give some proportion of salicylate in sufficient amount to lower the temperature

and keep it as near the normal point as possible. This applies not only to the acute stage of the infection, but to that salubrate condition often falsely regarded as convalescence. In most cases 100-200 grams will be an adequate daily dose of salicylate.

We must next inquire as to what are the imaginary and what are the real dangers involved in the use of salicylate. In former days, many of the bad effects of the drug were due to its contained impurities, but there is now no difficulty in acquiring the artificially prepared salicylate in a high state of purity.

Headache and tinnitus, which are so commonly listed as *adverse*, are in children hardly ever produced by the administration of salicylate. The former, indeed, is a frequent symptom of rheumatism, and is quickly alleviated by this treatment. Cardiac depression is a symptom constantly attributed to salicylate, but both clinical and recent experimental evidence contradict this statement (p. 12). On the



FIG. 12. EFFECT OF A RHEUMATISM TREATMENT ON THE PUPAL HEART.

During the first 10 minutes there is a period of cardiac depression (note the fall in the pupal heart rate). During the next 10 minutes the heart rate is normal (note the rise in the pupal heart rate). During the next 10 minutes the heart rate is normal (note the rise in the pupal heart rate). During the next 10 minutes the heart rate is normal (note the rise in the pupal heart rate).

other hand, it has been clearly proved by Dr. Lees and others that cardiac dilatation is an almost constant result of acute rheumatism. Indirectly, cardiac depression may be produced by the drug if it be allowed to upset the digestion, but probably in no other way. Anaemia, and even albuminuria and haematuria, have been regarded as due to the administration of salicylate, but these again are the effects of the disease, and not of the treatment. Mental depression is another rheumatic symptom (p. 166), and is only the result of salicylate when the drug is allowed to produce nausea or vomiting.

The real dangers of salicylic-acid poisoning are those of acid intoxication, vomiting, air-hunger, acetonaemia, delirium, and coma which may end fatally. These have been fully discussed in the section dealing with acid intoxication (p. 82), where the methods of avoiding them by the prevention of constipation, and by giving alkalis in doses

sufficient to keep the urine alkaline, have also been described. Dr. Lenz has shown very conclusively that, if thought desirable, very large doses may be given with perfect safety. The largest daily dose ever given to a child is, I believe, two grains, and this was unaccompanied by any symptoms of poisoning.

If we are to use salicylate to its best advantage, we must get rid of the dread of the imaginary dangers imputed to it, and substitute for them the real danger, that of acid intoxication. Further, we must be prepared to give the drug in larger doses—say to one or two hundred grains daily—than are often ordered, our object being to keep the temperature as near normal as possible.

If we wish to give the salicylate in these moderately large doses, we must adhere to the following plan. Starting with small amounts, the dose is rapidly increased by 30 or 50 grains per diem, care being taken that the bowels are well opened before an additional amount of the drug is given, and that the urine is rendered alkaline by an equal or a double quantity of the bicarbonate or citrate of soda. Should the child vomit, the drug should be stopped for twelve or twenty-four hours, and then restarted in half-doses, and rapidly increased. It is usually found that after this a dose equal to or greater than the amount which previously produced vomiting will be well tolerated. As the bulk of the dose is quickly excreted by the urine, it is well to give frequently repeated doses. A convenient plan is to administer ten doses in the twenty-four hours, two-hourly by day and four hourly by night.

The treatment of the symptoms of acid intoxication is given on p. 84.

Aspirin is less quickly absorbed and more slowly excreted than is sodium salicylate, and has the serious drawback that it cannot be administered in combination with alkalis.

It must be remembered that the condition of the heart may call for symptomatic treatment, and cannot always be treated by salicylate alone.

The alkali given with the sodium salicylate not only tends to prevent the danger of acid intoxication, but has a tonic effect upon the heart, possibly partly by neutralizing the acid products of the rheumatic organism. It also tends to play the gastric irritation which may be caused by salicylate. Glysterin or syrup of ginger or orange may be added to the mixture in order to render it palatable to children. It is an unpleasant mixture however given, but it is astonishing how well children take it, and how soon they become accustomed to it.

In the treatment of acute rheumatism no drug is comparable to the salicylates. Quinine and potassium iodide have a slightly beneficial action. Aspirin seems so far as I have watched these effects, appear to be at present of no value in the treatment of the infection.

CHOREA.

Definition.—*Acute or Sydenham's chorea* is a disease in which there are disordered muscular movements associated usually with some degree of muscular weakness, and very constantly with slight or profound mental disturbances. Is it the same cardiac changes and the same recrudescences and relapses that occur in acute rheumatism, are very prone to develop.

Etiology.—By defining the disease termed chorea in this way, we are enabled to exclude a variety of conditions which, although running a very different clinical course, show disordered movements resembling those of true chorea.

The etiology of chorea may be most conveniently discussed under two headings: those of the rheumatic or essential factor, and the neuropathic or predisposing factor.

Rheumatic Factor.—It has long been recognized that there is a very close association between chorea and the rheumatic infection. I use the term "rheumatic infection" in order to emphasize the important fact that we are not here concerned merely with a disease of joints, but with an infection of which arthritis is only one and often an unimportant manifestation.

Many series of figures have been given in connection with this question by various writers. Those founded upon 200 consecutive cases of chorea taken from the records of the Paddington Green Children's Hospital, agree in the main with those from other institutions.* These may be tabulated thus:—

Had previous joint rheumatism	99 per cent.
<i>(b) the remainder:—</i>	
Developed joint rheumatism during chorea	1 per cent.
Developed systolic apical bruit during chorea	91 —
Developed double apical bruit or severe heart lesions during chorea	1 —
Total	27 per cent.

These figures, it will be seen, exclude all cases in which the only evidence of rheumatism were tonsillitis and muscular pains. Neither do they include cases which develop rheumatic arthritis at a date subsequent to that of the chorea, a class of case which, as Dr. Batten has shown, is by no means uncommon. Further, the large group of cases in which there is dilatation of the heart unaccompanied by any apical bruit is not represented. We see, therefore, that there are numerous cases showing evidence of being rheumatic with which we

* For the statistics given in this article, I am much indebted to Dr. M. Fraser, who collected the figures from the records of 200 consecutive cases of chorea admitted to Paddington Green Children's Hospital.

might increase the percentage given above. It must also be allowed that it is possible for chorea to be not merely the first, but even the only recognisable evidence of the rheumatic infection. Such cases are, however, very exceptional, for it is a matter of every-day experience to be able to trace the presence of other rheumatic manifestations in the subjects of chorea.

Lastly, we can, I think, gain much information on the subject of the close association between the rheumatic infection and chorea not only by examining chronic children for evidences of rheumatism, but by looking in rheumatic children for symptoms of chorea. This question is referred to under LATENT CHOREA (p. 165).

The etiological factors of rheumatism have therefore a bearing upon chorea. The age-incidence of chorea follows that already given for rheumatism. Considering admissions to hospital for first attacks of chorea, it is found to be uncommon under the age of five years, only 4.2 per cent of the admissions being under that age; it becomes frequent between the ages of seven and twelve, being most common (19.15 per cent) between the seventh and eighth birthdays. The seasonal incidence shows that there is a slight increase in chorea during the winter months, 52.9 per cent of the admissions for chorea being during the five months of September to January. Other infections predispose to chorea; post-scarlatinal chorea is by no means a rarity, while diphtheria and measles are less often quickly followed by chorea. A family history of acute rheumatism, taking only into consideration parents and brothers and sisters, is very common in chorea.

Neuropathic Factor.—Given the rheumatic infection, the less stable the patient's nervous system, the greater is the liability to the development of chorea symptoms. Many points demonstrate this. The *sex-incidence* shows that, although articular and other forms of rheumatism are about equally frequent in both cases, chorea is between two and a half and three times as common in girls as in boys. The usual subject for chorea is the bright, intelligent and excitable child, one capable of working too hard at school. Anything that helps to render the nervous system unstable predisposes to chorea in a rheumatic child. Thus unduly hard work or excessive worry at school are common conditions to which chorea is attributed. Fright, again, is given as a cause of chorea in about 12 per cent of the cases. In both these matters, however, it is necessary to see the evidence carefully, for it is often not a case of cause and effect. In the majority of instances it is because the child has already got chorea, that its schoolwork is too hard for its restless, inattentive brain. Or again, because the child is already slightly choreic, it is frightened by something that would not have had any effect upon it had it been in good health. There is no doubt that both overwork and fright can make chorea much worse, and thus may produce severe symptoms in a case previously so slight as to escape recognition; but it is extremely doubtful whether either can of themselves originate an attack.

A neuropathic family history is not uncommon in cases of chorea. Chorea itself is present in the parents, or brothers and sisters in about 14 per cent of cases. The relationship between chorea and epilepsy is of interest. The child of an epileptic is prone to develop chorea if infected by rheumatism; while on the other hand a certain number of choreic children develop epilepsy at a later date. It is a peculiar fact, however, that an attack of chorea seems never to bring on or be synchronous with an epileptic fit.

In the consideration of the rheumatic and neuropathic aetiological factors of chorea, I have termed the former the essential factor. In this connection two questions require attention. First, is chorea invariably of rheumatic origin? and second, is chorea always associated with an active rheumatic infection?

Is chorea invariably of rheumatic origin? From its nature, this question does not admit of an absolutely certain answer. It is undoubted that choreiform movements may be seen in children in conditions which have nothing to do with rheumatism. They are found in some cases of cerebral diplegia, hysteria, thalamic lesions, rarely in tuberculous meningitis, and some instances of ischaemic poisoning; but with such different clinical courses and tendencies, such cases as these should not be classed under the heading of chorea. It is now held by many, and with this view I am in complete agreement, that chorea is always rheumatic in origin. Although this cannot at the present time be absolutely proved, yet by statistics and by daily experience, the association between chorea and rheumatism can be shown in such a large majority of instances, that it becomes most highly probable that this post-rheumatic view is correct. In the small number of exceptional cases in which no rheumatism can be traced, it is far easier to imagine that the chorea is the only manifestation of rheumatism, or that the other symptoms have been overlooked or forgotten, than to suppose that the disease has originated as a pure necrosis without any rheumatic origin. On the latter theory, the cases which appear to be non-rheumatic should be common, whereas we know that they are very exceptional.

Is chorea always associated with an active rheumatic infection? While we may reasonably hold that every case of chorea has its origin in such an infection, we cannot suppose that such bacterial activity continues or necessarily throughout the whole course of the disease. It seems certain that all the symptoms of the chorea may continue after the activity of the infection has died down, being now dependent upon the damaged condition of the nerve-cells resulting from the preceding infection. The absence of any regular nocturnal fever, the lack of response to treatment by salicylates, the beneficial effect of antineurotic and educational methods, all suggest that, in the later stages of chorea, the symptoms are dependent upon damage done by a past infection rather than upon a present bacterial activity.

Morbid Anatomy.—Drs. Poynton and Holmes have described the presence of streptococci and cell-infiltration in the perivascular spaces of the cerebral vessels, with engorgement of the pso-stachoid. They found that the nerve-cells showed advanced toxic changes. From clinical evidence, to be given later (p. 164), we may suppose that in certain cases the changes are not confined to the cerebrum, but may extend throughout the central nervous system.

There is some evidence to show that in the slighter forms of chorea the damage to the nerve-cells is due to a circulating toxin rather than to an actual bacterial infection of the nervous system. Probably in no case is chorea truly "infectious."

The cerebrospinal fluid is usually increased in amount. In most cases no excess of cells is demonstrable, but in the severer forms of the disease such an excess is present. Diplo-streptococci have been found in the fluid in a number of instances, but are absent in many cases.

Symptomatology.—Chorea is much more than a disease in which certain disordered movements occur. In every case examination should be made for mental, motor, and toxic symptoms. These will be described first, and followed by some mention of residual and latent chorea.

The term *hemichorea* is best not used, as it is inaccurate. The movements are never entirely unilateral and the mental symptoms, which are constant, are excluded by such a name.

Mental Symptoms.—Serious mental disturbance is always present in chorea, and thus the mental symptoms are more constant than the motor phenomena.

During the prodromal stages of the disease, signs of nervous instability, such as undue fearfulness, irritability and excitability, make their appearance, or where naturally present, become more marked. With these are often associated headache, sore throat, and pains in the limbs or sides. Upon the signs of nervous instability of rheumatic origin may be grafted such conditions as light-sensitiveness, occasional nocturnal convulsions, night-terrors, somnambulism, and hiccoughs.

As the choreic movements increase, the mental symptoms become, as a rule, more definitely those of depression. The child becomes very unhappy, and cries at the slightest word of reproof, while the fears, outbursts of passion and undue excitability increase in their severity. Incontinence is common, and is not seldom a very troublesome symptom.

While these are the usual mental symptoms, they may be much more severe, and occasionally they quite overshadow the clinical picture of the disease. They may be of the nature of melancholia or stupor.

The melancholia is occasionally so severe that suicide is threatened.

or even attempted; but this is rare. A condition of agitated melancholia, often spoken of as choreic mania, may be seen. In this, the patient is acutely insane, appearing in a violent and prolonged delirium. With it, the extreme restlessness and loss of sleep may produce severe and even dangerous exhaustion. It is less common in children than in adults.

The tendency to stupor is often seen in severe cases. Usually it does not go beyond a condition of drowsiness, but in one group of cases it proceeds to absolute dementia. This type is of interest, because it may give rise to a fear of a fatal ending. Its characteristics are these: The most noticeable feature is perhaps the extremely poisoned appearance of the patient. So marked is this, that the faces almost suggests that of a case of typhoid fever. The face is thin and anxious-looking, pale, with some flush on the cheeks. The lips are cracked and covered with sores, the tongue is furred and dry. The mind is apparently completely blank. There are aphasia, great weakness, rapid wasting, but practically no movements. Usually the case conforms to the paralytic type. The abdominal reflexes are absent, and there is incontinence of both urine and feces. The heart does not show the severer forms of inflammation. Although the patient appears extremely ill, recovery is the rule.

The memory is affected in all severe cases, but there are others in which the loss of memory is more marked than the other symptoms might lead one to imagine. Delayed combustion is usually present in chronic children.

The crying of choreic children needs a moment's consideration. As has been mentioned in the early stages, and indeed throughout the course of the disease, crying is easily caused. But in the severer cases, what appears to be spontaneous crying, the outcome of the mental depression is very common. It is likely to be associated with a good deal of snoring, owing to the irregularity of the respiratory movements in the disease. In such cases a very characteristic sound is heard which is only to be likened to the howling of an animal.

During convalescence the mental symptoms are those of exaltation rather than depression. The child seems extremely happy, and is usually a universal favourite in a ward. There is undue excitability.

Motor Symptoms.—In their earliest stages these consist of fidgety movements, which can at first be controlled momentarily by the patient. They are confused with some clumsiness of voluntary movement, and are often best appreciated when the child is endeavoring to make use of its hands, as in sewing, or doing up buttons. They are increased by emotion or excitement.

As the disease becomes more definite, the general motor restlessness increases, and the choreic movements become unmistakable in the face, limbs, and trunk. They are now to a large extent uncontrollable, and usually become worse under observation. In marked cases the

movements are perpetual, and an extraordinary degree of restlessness is apparent.

When the movements are severe, they are likely to be associated with or followed by considerable muscular weakness. The motor symptoms, in common with many of the mental phenomena, tend to disappear should severe cardiac symptoms arise, and to reappear when the heart improves.

The facial movements are necessarily of the emotional type, and expressions of pleasure, pain, surprise, attention and others are constantly fitting across the face. The commonest is perhaps the rapid and unconvincing smile of welcome, a veritable "society smile," which appears on the child's face on the approach of a visitor. In mild cases these facial movements express, or rather over-express, the mental emotion of the moment, but in the severer forms of the disease it is probable that they are not associated with any psychical feeling. In addition, there are grinning movements of the mouth and clicking sounds produced by movements of the lips and tongue. In bad cases, the tongue, when protruded, is held between the teeth and curled up towards the nose. In a few of the atonic cases the eyes assume a slanting direction, giving the face a slightly Mongolian appearance.

The movements of the limbs are so well known as to render description needless. They consist for the most part of perpetual twisting and bending movements; but it is to be noticed that there is a good deal of ataxia of the limbs present. This may be seen in the voluntary movements of the arms, and is often very obvious in the gait of the patient. The most characteristic point about choreic movements is that they are unsustained; what will be the next movement cannot be foretold. The movements may be more marked in the limbs of one side than in those of the other, but can never be said to be entirely unilateral, owing to the involvement of the muscles of the face and trunk. When held out, the hands usually show some hyperextension of the fingers. An extreme instance of this is seen in *Fig. 33*.

In the trunk, chorea produces squinting movements. In cases of any severity, what is known as dissociated breathing is seen. It consists of irregularly alternating thoracic, abdominal or normally contained respiratory movements. Long-drawn sighs are common.

Alterations in speech are largely due to the inco-ordinate working of the labial, lingual, laryngeal, and respiratory muscles. Usually the speech is delayed, and the words are indistinctly and explosively uttered. In some there appears to be present an element of scanning speech. Occasionally, the tone of the voice acquires a markedly nasal character, but I have never been able to demonstrate any definite weakness of the palatal muscles. The most severe speech defect is that of aphasia. This may remain absolute for many weeks or months, but invariably clears up entirely. It may persist for as long as nine months. It is usually associated with the severer atonic cases.

Ocular symptoms are sometimes present. The pupils show various signs. Dr. Lainghead has described rhythmical oscillatory movements of the iris (hippus), and inequality and eccentricity of the pupils. In my experience, such changes as these are very common, particularly during the earlier stages of chorea. The displacement of the pupil, I think, is always upwards and inwards. The reaction to light and accommodation is usually brisk but ill-sustained; to the latter, however, it may be sluggish or apparently absent. The pupils are very commonly dilated. Rarely, there is a departure from the normal circular form of the pupil. I have seen a pupil remain oval in chorea for several weeks. Slight nystagmoid movements in the eyes are very



Fig. 1.—CHOREA. FINGER-EXTENSION OF FINGER.

common; but well-marked nystagmus is quite rare, although it certainly does occur. I have watched it appear and disappear in two attacks of chorea in the same child.

The reflexes in chorea require mention. The tendon-perks are difficult to elicit, but are usually brisk when obtained. Some cases show what is known as the choreic or sustained knee-perk in which the extensor muscle is thrown into spasm, and the response to the tap on the ligamentum patellæ is unduly sustained. In other cases a double response is seen. Similar changes are found in the tendon reflexes in the arms and at the ankles. A suprapatellar perk can not uncommonly be elicited. The knee-perks are occasionally absent; but the more patience that is exercised in examining these reflexes, the fewer will be the failures to elicit them. In some cases of the severe atonic class, they appear to be quite absent. The abdominal

reflexes are usually very brisk; the cases in which they disappear have already been described (p. 161). In the same group, continual double incontinence is present, but in the usual case of chorea there is no more than an occasional incontinence of urine. The pupillary reactions have already been mentioned.

Sensory symptoms are very uncommon in chorea. I have found in a few patients diminished sensibility or anaesthesia of the pharynx.

Atonic Symptoms.—In most cases of chorea there is some muscular weakness present. It shows early in the diminished power of the hand-grips and in the muscles of the neck. In more severe cases the muscles throughout the body are weak and flabby (*atonia musculi*), but in the worst they are entirely powerless (*atonia paralytica*). Such paralysis usually follows the stage of movements and may be associated with rapid wasting of the limbs and with aphasia. As a rule some power of movement of the fingers remains. The weakness may be more marked on one side of the body, but generally a demonstrable paresis is present on the other side.

General Symptoms.—The temperature in the rheumatic infection has been fully described (p. 150), and the fever in chorea is of a similar type. At first wholly above normal, the temperature becomes swinging and intermittent in character. It is to be remembered—*a point already discussed*—that the choreic symptoms may outlive the activity of the bacterial infection, and being now dependent upon the resultant damaged state of the nerve-cells, will run an afebrile course.

Other rheumatic manifestations may exist with chorea, of which the chief are endocarditis, heart-aneurysms, and anaemia. Rheumatic pains or arthritis are seldom seen together with well-marked choreic movements, while the onset of severe acute cardiac dilatation is generally associated with a diminution of the motor symptoms of chorea.

The commonest cardiac change in chorea is slight dilatation, with which is associated a reduplicated and rather slipping first apical sound. Irregularity of the heart's action, due to irregularity of the respiration, is common. Sometimes it is extreme, and to this condition the term "*chorea cordis*" is given by some. It conforms to the type known as sinus arrhythmia. The more severe cardiac changes are those of the various forms of rheumatic inflammation.

The thyroid is enlarged in a small proportion of cases of chorea, most commonly in girls of ten to twelve years old. Such an enlargement, however, is not uncommon at this age in female children in the absence of toxæmia.

The symptoms of chorea have now been discussed, and it may perhaps be of interest to note that there is some evidence to support the view that chorea is not necessarily due only to a cerebral infection, but that similar changes, probably in certain cases, occur throughout the central nervous system. The ataxia, the suggestion of scanning

speech, the rate nystagmus, and possibly some of the loss of muscular tone, suggest that the cerebellum may be involved; while the rapid wasting accompanying the flaccid paralysis of the worst atonic cases, might be due to damage of the anterior horn-cells of the spinal cord. Such changes have not, however, yet been found in the cases which have been examined pathologically.

Residual Chorea.—This term is used for those cases in which symptoms of chorea remain after the acute stage of the disease has passed off. As a rule, residual chorea is due to the damaged state of the nerve-cells, and not to an active infection; but the possibility of the infection being merely quiescent and not destroyed must be borne in mind, and this is especially to be feared where *zoster* and a nocturnal rise of temperature persist.

The symptoms in residual cases are usually a combination of the mental, motor, and atonic symptoms already described. On the mental side the child is irritable, but no longer depressed. The movements are still present, and the voluntary actions are usually particularly clumsy and ataxic, while the muscles are very flabby.

Latent Chorea.—The symptoms of chorea when fully developed have been described, and we have here to deal with a condition which could hardly be recognized as one of chorea unless we are able to trace that disease back to its earliest symptoms. It is, however, a matter of frequent experience to watch the development of chorea.

The rheumatic infection, when of sufficient severity in proportion to the nervous stability of the child, produces the group of symptoms which we term chorea; but long before this point is reached it has caused a series of milder symptoms denoting a general nervous instability which we may term latent chorea.

In latent chorea the child is pale, fidgety and, in the finer movements of the limbs, clumsy. He is nervous, easily frightened and upset, affectionate but passionate when crossed, excitable but readily exhausted, unable to concentrate his attention on anything for more than a moment, and hence getting into trouble at school, particularly over his sums. Such symptoms are of course merely indicative of nervous instability, and not of necessity related to a rheumatic infection; but the point to emphasize here is that they may be, and at London very commonly are, due to such an infection, and may then legitimately be regarded as latent chorea. In these cases there may be such manifestations of rheumatism as sore throats, pains in the limbs or sides, headaches, evening fevers and dilatation of the heart. Further, as the result of the nervous instability which is in these cases of rheumatic origin, there may arise such nervous disorders as habit-spasms, night terrors, somnambulism, acquired enuresis, hemeric diarrhoea, and the like.

The disorders named, and the state of general nervousness described,

have for long been noted as particularly prone to be present in rheumatic children, and it has become vaguely held that rheumatism is peculiarly liable to attack nervous children. While it is true that chorea is more liable to occur in a neurotic than in a normal child, if it acquires rheumatism, it seems impossible that a nervous child is particularly likely to be attacked by a certain bacterial infection, whether it be rheumatism or any other. It is easier and more accurate to suppose that the association of general nervousness and various nervous disorders with rheumatism is due to the fact that the child is already mildly infected with the disease, and that the nervous instability is the result and not the cause of such an infection. It is to this state of nervous instability, when due to rheumatism, that we may apply the term *latent chorea*.

It is probable that latent chorea is due to a rheumatic toxæmia and not to a rheumatic infection of the nervous system. This is, however, a point of little importance as compared to the fact that it is of rheumatic origin.

Latent chorea may be seen in three groups of cases: (1) *In children with obvious acute rheumatism*; (2) *In children convalescent from acute rheumatism or chorea*; (3) *In children without any very obvious rheumatic symptoms*. In all three classes the symptoms of latent chorea are very similar. We are here, however, only concerned with the first two groups.

1. **Children with Acute Rheumatism** of the joints, muscles or heart usually show that the nervous system has not escaped. If chorea be recognized at its earliest stages, it is certainly true that nearly all rheumatic children have chorea. The mental symptoms are those of depression. This is not always due to the pain, which is usually very slight when the child has been put to bed, and it is certainly not always due to the treatment by salicylate, for it is present where none has been given. There are also undue timidity and general nervousness in most cases. Some fidgety movements are present, which cannot be detected by the ordinary test of making the child hold its hands out in front of it; but they are clearly seen going on beneath the bed-clothes as one approaches the patient, and are brought out during the voluntary movements of the hands, which are jerky and clumsily performed. They can often be appreciated well when the pupils are being examined, for the eyes will not remain fixed in any position for more than a moment. The pupillary changes which have been described under chorea are often present in cases of acute rheumatism, as Dr. Langmead has noted.

This type of latent chorea has been described by many authors, sometimes under the name "*sub-chorea*."

The recognition of chorea in cases of acute rheumatism adds another link to the chain of evidence binding rheumatism and chorea as the result of one and the same infection.

2. **During Convalescence from Acute Rheumatism and Chorea** much

the same symptoms are observable save that the depression has now given place to mental exaltation. The child is very happy, full of life, everybody's favourite in the ward. He is likely to be unduly excitable; he is obedient, affectionate, and sharp. He is no longer shy, but is very willing to become your friend. He is apt to take liberties, and in his excitement may be very rude in a glib way, but is quickly subdued to fears of punishment when reproved by one in proper authority. During this stage the "society smile" of welcome, already described, is very much in evidence, and is very characteristic.

3. **Latent Chorea without Obvious Signs of Acute Rheumatism** is fully discussed under the etiological factors of functional nervous disorders (Section X).

Diagnosis.—There is seldom great difficulty in recognizing chorea. The acutely delirious cases resemble some instances of pneumonia, typhoid fever, and atropine poisoning. Pneumonia is a much more common cause of delirium in children than is chorea. Choreiform movements may be seen, as has been mentioned in other diseases, of which the most common is cerebral diplegia. In this, however, the movements are life-long and the face is not affected so that a consideration of the clinical history of the case renders the diagnosis plain. Chorea paralytica is usually easily recognizable by the emotional facial movements which are still present. Further, there is a history of previous movements in the limbs.

The greatest difficulty arises in connection with very slight cases, and here it is well to rely upon other rheumatic manifestations. Where these are present, the likelihood of the signs of nervous instability being due to chorea becomes marked. The differential diagnosis between chorea and habit-spasm is given elsewhere, but it is to be remembered that a habit-spasm may be engrafted upon a latent chorea. In some cases it is extremely difficult to say if a child has chorea or is mentally backward, with the fidgeting which is so commonly seen in such a case. Here the diagnosis may rest entirely upon the previous history and the later course of the disorder. It must be borne in mind, however, that a mentally deficient child may be infected with rheumatism and develop chorea.

Course and Prognosis.—Chorea by itself practically never causes death. In the 300 cases already quoted, no death was attributable to the chorea. Very rarely, severe motor symptoms cause death from exhaustion and loss of sleep. The severe mental cases, although appearing dangerously ill, do not die. The cause of death in chorea is cardiac thrombosis, usually pericarditis. From such the mortality is in Dr. Fraser's figures 1.5 per cent.

The course of the disease is seldom less than four or six weeks, and may be protracted for months. A poor appetite, anemia and insomnia

are symptoms which lengthen the attack; but of greater importance in this connection are muscular weakness, paralysis, and aphasia. It must, however, be remembered that the pathetic symptoms may develop in a sthenic case. No case should be looked upon as of slight severity in its early days, for a sudden development of severe symptoms not infrequently occurs; more particularly is this the case with the mental symptoms.

The tendency of the disease is towards complete recovery without permanent damage to the brain. The alarming mental symptoms, even long-lasting aphasia, clear up entirely in time. Relapses and recrudescences are, however, common.

Treatment.—As in other diseases, there is no routine treatment suitable to all cases. The antirheumatic, the antineurotic and the sedative forms of treatment, each has its place; but at the same time it must be remembered that the real danger of the disease lies in the rheumatic affection of the heart.

On the first signs of chorea the child should be put to bed, and is better attended by a nurse than by a member of the family. In mild cases, all forms of quiet amusement should be allowed and are indeed beneficial, but anything raising excitement, as is easily the case, is harmful. In severe cases strict isolation with nurses is very necessary, and precautions must be taken to prevent injuries to the child. A good plan is for a bed to be made up on the floor in the corner of the room, the walls being padded by mattresses. Failing this, the child's limbs may be strapped up, especially the elbows and knees; or if in a cot, bolsters should be placed along the sides of the cot.

The diet should consist, in acute cases, of milk, milk foods, eggs, and chicken, given in as large quantities as the constitutional condition of the child allows. In many cases the patient will have to be fed by the nurse, and it may be safer to make use of thick drinking utensils that will not break if bitten. In convalescent or residual cases, it is best to give an ordinary varied diet, to which extra quantities of milk, cream, butter-fat, and eggs are added; the child should be given as much as can be digested.

Of drugs, salicylates hold the first place, because they treat the cause, and not only the symptoms, of the disease. In any case which shows signs of an active rheumatic infection, salicylates should certainly be given. Probably, bearing in mind the tendency to recrudescences, it would be best to give all cases a course of such treatment. The result of salicylate on the chorea itself has been held by some to be disappointing, although in many cases it is, they say, certainly beneficial. To this objection it may be answered, firstly, that if we can check or ward off cardiac mischief, we are doing more good than by merely damping down choreic movements; secondly, that in some cases the symptoms are so large due to an active rheumatic infection, and are naturally not benefited by salicylate; and thirdly, that to obtain the

best effects in chorea, the drug should be given in doses rather larger than those ordinarily administered (100-200 grains daily). Salicylate should certainly be given where there have been recent rheumatic pains, or where there is a regular rise of temperature above the normal, even though this is only seen at night. Theoretically, it may seem unsound to awaken the child at night for a dose of medicine, but in practice it is found that the patient quickly falls asleep again.

Of sedative measures, none is better than the hot pack, and this may be combined with the treatment by salicylate when necessary. Blankets should be wring out in very hot water, and the child rolled up in them, surrounded by eackintosh sheeting, which is covered with warm, dry blankets. This rarely fails to produce sleep within a few minutes, and the patient may be left in the pack while asleep, being dried with warm towels on awaking.

Of sedative drugs, trional, chloral and bromide are the most useful. Of these I should be inclined to give the first place to trional, as recommended by Dr. Voelcker. It may be given as a powder to a child of ten, starting with five-grain doses every six hours, which may be pushed to ten grains four-hourly. It should not be continued in large doses for more than a week, for although I have never seen any such result, the possibility of leucotopoeophrynemia must be borne in mind. Chloral and bromide (five grains of each four-hourly) are useful sedatives, but are not so successful in my judgment as trional. They are, however, if carefully used, practically free from danger. Sulphonal is too dangerous a hypnotic for the treatment of chorea. I have not been struck with the good effects that have been claimed for chloroform in this disease. Brandy may be used alone, or in combination with other measures, to promote sleep at night.

In atonic or aphasic cases, the sedative drugs are harmful and should not be given.

Most authorities are now agreed that the very large doses of arsenic formerly advocated are too prone to cause gastro-enteritis and peripheral neuritis to render their use justifiable. Ergot has been recommended by Dr. Eustace Smith.

During convalescence the antineurotic line of treatment is the most successful. A very full diet should be given, and may be combined with tonic drugs. Cod-liver oil, iron, arsenic, even strychnine are of much service. The flavoured brand of sanatoxes, given in half-drachm doses at milk three daily, is of considerable value at this stage. Where there is much muscular flabbiness, massage is very useful, and may be combined with tepid or cold sponging, or douching.

Of great value in convalescence is the re-educational method. The child is encouraged to control the movements of the limbs by the use of various games, and by kind, firm discipline. If the condition of the heart allows, regular walking exercise may be taken, the child being encouraged to conquer the atonia which will probably be troublesome at first.

As soon as it can be managed, a visit to a bracing country place should be arranged. The child should not be sent back to school for at least six months, and even then the effect of it should be carefully watched. Hospital children, however, are often less worried at school than at home, and may return more quickly. Simple home-treatments, quietly but regularly given, should be undertaken, and every effort made to rebuild a physically and mentally healthy child.

IV.—RHEUMATOID ARTHRITIS

Rheumatoid arthritis in children is usually, as was pointed out by Dr. Still, associated with enlargement of the spleen and lymphatic glands. This fact, taken in conjunction with the clinical course of the disease in young subjects, makes it almost certain that the origin of the arthritis is locustærial.



FIG. 34.—ILLUSTRATING CHANGES OF RHEUMATOID ARTHRITIS. PHOTO A YOUNG GIRL WITH RHEUM. SPLENOGAL.

Now that rheumatoid arthritis has been accurately defined and described by Dr. A. E. Garrod (*Trans. Med. Soc. Lond.*, 1907), we know that it does not vary much with the age of the patient. The enlargement, however, of the spleen and glands, which is exceptional in adults, is the rule in children, in whom also the acute forms of the infection are particularly conspicuous.

Etiology.—Girls are rather more frequently affected than boys, in the proportion of three to two. When it occurs in children the disease usually makes its appearance before the age of six years. Occasionally it starts during infancy.

While it can scarcely be disputed that the disease is the result of an infection, we cannot as yet say to what organism it is due, nor indeed are we even in a position to assert on clinical grounds that it is the result of one organism alone.

There is some evidence which points towards the arthritis being the result of a streptococcal infection which may have its source in some local focus of suppuration in the mouth or elsewhere, in certain of the cases.

The relationship between rheumatoid arthritis and acute rheumatism is of considerable interest. In both conditions we find an infective disease of joints mainly affecting the synovial membranes and peri-articular tissues; in both we find anaemia, moist skin, a mild leucocytosis, nodules, and affections of the heart. The two latter, the most interesting points of similarity, differ very markedly in their characters in the two conditions. But the connection between the two diseases is seen in other ways as well. Certain examples of rheumatoid arthritis start so acutely, that at their onset they cannot be distinguished from acute rheumatism. Further, in some cases we find rheumatoid arthritic changes following a series of attacks which appeared to be of the nature of acute rheumatism. Lastly, in rare cases the joint changes of rheumatoid arthritis are found to be associated with valvular heart-disease, although there may be a complete absence of any history of acute arthritic attacks. Fig. 34 shows a hand of a young woman who had chronic arthritic changes typical of rheumatoid arthritis, together with a typical severe mitral stenosis. She had never been laid up in bed with any joint trouble, so insidious was the onset of the arthritis.

The connection between rheumatoid arthritis and acute rheumatism is seen to be very close, although in typical cases of the two conditions there are many points of profound difference. Two explanations are possible. We may hold that the organisms of acute rheumatism may occasionally and atypically produce rheumatoid arthritis, or we may regard a double infection by the organisms of acute rheumatism and of rheumatoid arthritis as a not unknown event. While it seems highly probable that the organisms of the two conditions are very closely allied, it appears to me that the connection between rheumatism and rheumatoid arthritis in certain difficult cases is best explained by assuming a double infection.

The cases which are said to follow repeated attacks of acute rheumatism are very rare in children, as are those of rheumatoid arthritis of insidious onset, but accompanied by valvular heart-disease. They require mention, however, in dealing with the etiology of the disease.

A rare form of chronic arthritis associated with inherited syphilis appears to resemble osteo-arthritis rather than the condition under consideration.

Symptomatology.—An insidious onset is very exceptional in children. In them the disease starts with a definite acute and febrile stage, and throughout its course it shows well-marked alternating periods of activity and quiescence.

The spleen is usually enlarged sufficiently to become palpable below the costal margin. Its enlargement is proportionate to that of the glands. The glands in relation to affected joints are nearly always enlarged. The suprasternal and axillary glands are those most commonly affected.

During the acute periods of the disease, the temperature is raised, and the joints become swollen and painful. The wrists, knees, and

fingers (Fig. 35 and 36) are those which are chiefly affected, the involvement of the last not being severe until late in the disease. The fusiform shape of the joint swellings is similar to that seen in adults. There is no evidence of osteophytic growth, and no bony sequestra are obtained. The spine may be affected. During the acute stages the ends of the bones in the affected joints are seen in a skogram to be indistinctly translucent. The nodules of this disease differ from those of acute rheumatism in that they are larger, harder, and more chronic. Further, they are often very tender during the acute stage, and their localization and lack of symmetry forms other points of difference from the rheumatic nodules. Enlargement of the bones



Fig. 35.—Enlargement of knee joint during acute stage of disease.

may be present. The skin of the face is moist and shiny, and not uncommonly freckled. The blood shows some diminution of the hæmoglobin, while during the acute stages there may be found a leucocytosis of about 18,000, a differential count of the white cells showing no striking abnormality. The anemia and discoloration of the skin are useful guides to the progress of the infection, where they remain bacterial activity is to be feared. The heart shows little change clinically. Persistent tachycardia is not uncommon. Occasionally, signs of chronic pleurisy may be detected at the bases of the lungs.

During the quiescent stages, the swelling and painfulness of the joints lessen, but contractures and muscular atrophy develop. For this reason the disease appears more regularly progressive than it really is. The atrophy of the muscles and of the skin is rarely severe during childhood.

Morbid Anatomy.—The joints show changes similar to those seen in rheumatoid arthritis in adults. There is thickening of the capsule of the joint and of the peri-articular tissues. The synovial membrane is somewhat thicker and more vascular than normal. The cartilages may show no change at all, but in most cases there is pitting of its surface, with small processes of the synovial membrane filling the minute pits. There is a complete absence of metaplastic changes (fibrositis and chondrositis). The change seen in the bones by X-rays has already been mentioned. It is only present in the active stages of the disease.

A peculiar feature of most fatal cases is that the pericardium is tightly and universally adherent to the heart wall, but there are not as a rule any external adhesions. The valves of the heart are normal. Frequently there are signs of chronic pleurisy at the bases of the lungs.



Fig. 10. Rheumatoid Arthritis. Hands and forearms.

Diagnosis.—In some cases, as has been mentioned, it is hardly possible to differentiate rheumatoid arthritis from acute rheumatism at the onset of the attack. Its nature becomes obvious as the disease progresses. In most instances, however, there is little difficulty in recognizing the disease at any period of its course. The possibility of the syphilitic chronic arthritis must be borne in mind. A non-syphilitic osteo-arthritis, similar to that seen in adults, is of excessive rarity in children.

Prognosis.—In children the disease rarely runs a favorable course, and with repeated severe exacerbations and the gradual onset of contractures the child becomes bedridden after a few years, and dies of some intercurrent affection. Slight cases may recover completely. In others osteo-arthritis may supervene. The persistence

of anemia as in rheumatism, together with the shiny and bronzed appearance of the face, is an indication of further trouble.

Treatment.—This is not as yet very satisfactory. Perhaps the most successful measure to employ is Bier's method of passive hyperemia. This, as has been shown by the late J. H. Wells (*Trans. Pac. Soc.*, 1907), is analogous to the treatment by vaccines, but it is of course applicable to a condition due to an unknown organism. Working on the effect of Bier's treatment in tuberculous joint affections, he showed that each production of hyperemia in an affected joint is followed by changes in the opsonic index similar to those seen after a vaccine injection. This being so, care must be taken not to apply the method too vigorously, lest harm be done. During the acute stage the bandage placed above the diseased joint should be removed as soon as puffiness begins to appear in the limbs; it may be applied daily. As the more acute trouble begins to subside considerably longer and more frequent applications of the treatment may be made. If successfully carried out, the improvement is seen in all the joints, while the constitutional change for the better is also marked. Drugs are not of any great service. Gluconol carbonate is perhaps the most useful, and may be given three times a day, in capsules containing two grains to a child of six or eight years, and this dose may be considerably increased. Salicylates have but a slight beneficial effect. Aspirin relieves the pain to some extent. Where much pain is present, the joint, previously carefully washed and dried, may be painted over with tincture of iodine and covered by a linseed poultice, as recommended by Dr. Luff. The poultice is kept in position for twelve hours, and may then, if necessary, be renewed. Various forms of balneo-therapy are of value. Good results of treatment by thyroid extract have been recorded.

Possible sources of infection in the mouth or elsewhere should be treated.

During afebrile periods, a liberal diet should be given, together with cod-liver oil and iron. Massage is of great value in keeping up the tone of the muscles and in preventing contractures.

V—ERYTHEMA NODOSUM.

This disease, which was formerly regarded as a manifestation of rheumatism, is now usually looked upon as a separate infection. Dr. Louden, in Australia, has termed it "nodal fever." Although probably not rheumatic, yet it certainly seems to occur more frequently than coincidence will explain in children who have had rheumatism. Some on this account hold that the organism of rheumatism may produce

erythema nodosum, and view the condition as due to various bacteria, one of which is that producing rheumatism. It seems, however, preferable to regard erythema nodosum as a specific disease, and to explain its occurrence in some instances in rheumatic children as due to a secondary infection, to which the damaged state of the vessels would make the rheumatic subject peculiarly liable.

Occasionally, one case of erythema nodosum is quickly followed by another in the same family. It is twice as common in girls as in boys, and is most frequent in the first six months of the year. (Cautley.)

Symptomatology.—The symptoms are comprised of fever, sore throat, muscular and arthritic pains, and the characteristic rash. The constitutional symptoms are usually mild, but may be severe, and may be present for several days before the rash appears. The rash is very easily recognized. It consists of slightly raised red patches, which may be entirely free from pain or may be tender, even exquisitely so. These occur most commonly on the anterior surfaces of the legs, reaching up to the knees (Fig. 32). They are not rare on the extensor surfaces of the forearms, and may also be found on the cheeks. Rarely, the rash is more extensive, and spreads along the outer and posterior surfaces of the thighs on to the buttocks. These lesions quickly fade, going through the various discolorations of a bruise. During the first few days after the appearance of the rash fresh spots may arise. With the onset of the rash, the constitutional symptoms generally disappear.

Diagnosis.—This is easy when the rash has appeared, but impossible prior to its eruption.

Treatment.—Apart from rest in bed, which should always be observed, this is probably of little benefit. Where there is much



Fig. 32.—*Erythema nodosum.*

The rash is fainter on the left leg,—inflamed on the right.

brodifens, glycerin and belladonna may be painted on the painful areas. Many drugs, such as the salicylates, citrate of potash, and the calcium salts, have been thought to do good; but it must be remembered that the tendency of the disease is towards rapid recovery. In severe cases it must be acknowledged that no medicinal treatment is of avail in diminishing the activity of the infection. I have seen the fever and arthralgia practically uninfuenced by the administration of sodium salicylate pushed to a dose of 200 grains per diem.

VI.—INHERITED SYPHILIS.

As to nomenclature: of the terms used to denote the common form of the syphilitic infection in children, "inherited" is more accurate than either "congenital" or "hereditary." Some have confined the use of "inherited" to those cases in which the syphilis dates from the time of conception, and would by "congenital" mean those instances where the mother becomes infected during the pregnancy. These subtle distinctions are of little clinical value and are here disregarded.

Inheritance.—The question of the mode of infection of inherited syphilis is one which is full of difficulty. The various possible ways in which such an infection may occur are: (1) From a syphilitic father; (2) From a syphilitic mother; (3) From both parents being infected; (4) From the mother becoming infected during the pregnancy.

Of these methods the first ("sperm infection") was, until recently, thought to be the most common. With the discovery of the *Spirillum pallidum* a fresh difficulty has arisen in accepting this view, for it appears that the causal agent of syphilis, at all events in the stage in which we know it, is too large to enter a spermatozoon.

The questions, therefore, concerning the infection of the fetus, do not at present admit of a definite answer, nor is this a matter of great clinical moment, for we are not able to recognize any particular results of any one method of infection. Clinically, practically only one rule is clear, and even to this there are not a few exceptions. It is to the effect that the more recent the parental infection, the more severe are the manifestations of the inherited disease. This is shown by the course of events which commonly occurs. The first pregnancies after infection result in miscarriages, in which the length of gestation is gradually increased. These are followed by premature or still-births. Then, perhaps, a child is born alive, but dies soon after birth to be followed by one who survives and shows evidences of inherited syphilis. There are, however, exceptions to this course of events, and even an apparently healthy child may be born between two infected

children. In the case of Lewis, one child has been recorded to be infected, the other apparently healthy.

It may perhaps be excessive to emphasize the importance of obtaining an accurate account of the family history in cases where there may be a suspicion of syphilis. Only too often, students are content with learning of miscarriages without tracing the periods of pregnancy in which they occurred, or their relationship in time to the birth of the patient. To be of value, the history of miscarriages and premature births must be to some extent in line with the course of events sketched above. Undue emphasis is often laid by students upon the totally insufficient evidence of a miscarriage. The effect of infection occurring after the birth of one or two children, is usually very marked in the later generations.

Immunity.—Certain facts on this point are well known, and constitute Colles's and Proteta's laws. The first is to the effect that a woman giving birth to a syphilitic child, although herself apparently healthy, cannot become infected by her child. Proteta's law is the contrary proposition, that an apparently non-syphilitic child, born of a syphilitic mother, cannot contract the disease from its mother.

These facts are not in dispute, for only doubtful exceptions to these laws have been recorded, and these very rarely. But the explanation of them is as yet unsettled. For instance, in the matter of Colles's law there arise such questions as these: Is the immunity of the mother due to a latent infection? Is it due to antibodies which her tissues have manufactured in response to the organisms confined to the fetus (active immunity)? Or has she merely shared in the antibodies that the fetus has itself produced (passive immunity)? To these questions no definite answer can be yet given.

It appears clear that in this type of case the immunity, however obtained, is not always life-long. Definite instances in which an inherited syphilitic has acquired the infection in adult life, and of two attacks of acquired syphilis in one individual, are known.

Infectivity.—Extraordinary statements have been made as reference to the contagiousness of inherited syphilis, and it has been said to cause hundreds and thousands of cases. As a matter of fact, infection from inherited syphilis is a rare occurrence. No one, however, would wish to minimize the danger of such a possibility, and parents must be warned that their infants are a source of danger to healthy individuals. The contagiousness is chiefly connected with the early lesions of the mucous membranes and skin.

There is no clear evidence to show that inherited syphilis is ever carried on to the third generation.

Frequency.—On the subject of the frequency of inherited syphilis amongst London children, Dr. Seal gives some interesting figures.

He finds that of the children outpatients only 50 per cent were undoubtedly syphilitic, and that when doubtful cases were included, the proportion was only raised to 55 per cent.

As to the fatality due to this disease in its inherited form, trustworthy figures can be quoted. Syphilis is often a cause of death which does not appear in death certificates. Were this otherwise, the extent of the damage done by the disease would yet be greatly under-estimated, for the great numbers of miscarriages and stillbirths would all be excluded.

Age-incidence of Symptoms.—Manifestations of syphilis are only exceptionally present at birth; of these, the commonest are snuffles, wasting, and syphilitic pemphigus. As a rule, the infant appears perfectly healthy when it is born.

The great majority of the cases show manifestations of the disease before the end of the third month. Various observers agree in stating that symptoms start in about three-quarters of the cases within the first eight weeks of life, and about 90 per cent by the end of the twelfth week. For the first symptoms to appear after the first year is extremely rare; but in a very few cases, symptoms may not be noticed until the late manifestations occur at the age of six or seven years. Such are sometimes known as instances of *syphilis hereditaria tarda*. It is of course possible that the infantile symptoms have been overlooked or forgotten.

The early manifestations of inherited syphilis which are common are the following: Snuffles, laryngitis, rashes, swelling epiphyses, enlargement of the spleen, liver and testes, iritis, choriochoroiditis, and nephritis.

In late infancy and early childhood, may be found anemia, bowing of the skull, and depression of the nose.

In later childhood the following may be present: Hutchinsonian teeth, interstitial keratitis, deafness ("the Hutchinsonian triad"), gumata, perostitis, joint affections, juvenile general paralysis or tabes. At about the sixteenth year, fresh symptoms of the disease usually cease to appear.

SYPHILIS OF THE RESPIRATORY SYSTEM.

Snuffles.—The earliest symptom of inherited syphilis is in the majority of cases the condition still known by the homely name of "snuffles." Usually occurring in the first few weeks of life, it is occasionally present at the time of birth. Obstruction to the breathing is the chief symptom at first, and may become so severe as to prevent the child from either sucking or sleeping. This is due to an inflammatory swelling of the nasal mucous membrane. There may be little discharge from the nose, but this usually develops quickly. As the inflammation of the nasal mucous passes

on to necrotation and destruction of bone, the nasal discharge becomes mucopurulent, and may be bloodstained. Small pieces of necrosed bone are occasionally found in the discharge. Round the nostrils there is much soreness and redness. From the destruction of bone the bridge of the nose becomes depressed, and the characteristic "saddle-nose" may develop (Fig. 38).

In treatment, apart from the application of the ointment of the yellow oxide of mercury to the nostrils where there is excretion, local treatment is not as a rule necessary. In bad cases, however, Dr. Sutherland recommends the use of a lotion of one part of black-wash and three parts of lime-water, in the form of drops, to be directed into the nostrils.

Laryngitis.—This, when present, develops soon after the appearance of snuffles. The child's cry becomes hoarse. More than inflammatory swelling of the mucous membrane of the larynx is unusual, but in some cases actual ulceration of the vocal cords and the cartilages of the larynx is found. In rare cases, cicatricial stenosis may develop at a later date. Occasionally the pharynx shows ulceration.

Perforation of the Palate.—Perforation of the palate, either hard or soft, is occasionally seen in inherited syphilis. It is almost entirely confined to the later years of childhood.

Pulmonary Syphilis.—Various forms of syphilitic pulmonary disease have been found in still-born children, but are not of common occurrence in this country. Of these, the so-called pneumonitis alba is the best known. In this, there are in the lungs areas of consolidation which are white in colour, and consist of air vesicles full of epithelial cells in a state of fatty degeneration. This condition, as a rule, is bilateral and incompatible with life, but occasionally it is one-sided only, and may be survived for a few months. In such cases, considerable fibrosis occurs in the affected lung, and Dr. Still raises the question as to whether this is one of the causes of the unilateral pulmonary fibrosis in children, or the type which is usually regarded as a post-pneumonic condition. Such cases as I have had traced have always failed to yield a positive serum-reaction.

Gummata of the lung have been described in infants. They are extremely rare.

SYPHILIS OF THE SKIN, Etc.

Syphilitic Rashes.—These are present in the majority of cases of inherited syphilis. They usually appear shortly after the development of snuffles. Very occasionally, the child may be born with a syphilitic eruption already in evidence (syphilitic pemphigus). As a rule, the rash appears within the first three months of life, and only rarely after the age of six months.

Congenital Eruption.—*Syphilitic pemphigus* (the *hidraden syphilitic*) is the only rash of syphilitic origin that is ever present at birth, but it may not appear until a few days later. It is one of the least common of the syphilitic rashes. The lesions are bubble, vesicle or flaccid, which are surrounded by reddish-brown halos. Broad flat papules of the same coppery-tinted tint may also be present. The lesions are most commonly present on the palms of the hands and soles of the feet. They are often in addition to be found round the mouth, and in severe cases they may be generalized.

From non-syphilitic pemphigus neonatorum, the syphilitic condition has to be distinguished. The latter is found in wasted infants, may be present at birth, and affects the palms and soles. The non-syphilitic



FIG. 3.—CONGENITAL Eruption: LESIONS PRESENT AND FADING ON SOLES.

eruption is found in well-nourished infants, develops in the first few days of life, but is never present at birth and is not seen upon the palms and soles.

Later Eruption in Infancy. These may be macular, papular, vesicular, pustular, or scaly, but they have in common a flat, circumscribed, disc-like appearance and their coppery-tinted ("rose taint") coloration. Where the rash is most abundant, as on the buttocks, thighs and round the mouth, coalescence is prone to result in the formation of large anular areas with isolated lesions at their margins. On the palms and soles the lesions remain discrete.

The extent of the eruption varies much according to the severity

of the disease. It is most commonly found in the regions already mentioned. The vesicular, pustular, and bullous lesions are much less common than the macules and papules.

The diagnostic points chiefly to be relied upon in distinguishing the syphilitic from other eruptions, are their coppery-brown colour, and their presence upon the palms and sides of the neck in its all extensive.

Fissures, radiating outwards from the mouth or anus, are common during infancy. They may leave permanent scarring. Rhagades at the lips, to be of diagnostic value, should not be confined to the angles of the mouth, but should be definitely linear and radiate outwards from the upper and lower lips (*Fig. 38*).

Condylomata are frequently found round the anus or in the mouth. They are raised, flat-topped patches of about the size of a threepenny-piece. They are very liable to occur at any time during infancy.

Later Skin Affections.—These are uncommon. Condylomata may occur up to the ages of three or four years. Gemmæ of the skin have been described. Phagedenic ulceration of the face is a very rare manifestation.

Hair.—The hair in syphilitic children may be abnormal in two ways. Excessive growth of hair, usually dark in colour, may be present during the first few weeks of life. It is sometimes called the "syphilitic wig" (*Fig. 39*). There is nothing peculiar about it except its luxuriant growth, and such a condition is by no means confined to syphilitic children.

A more characteristic change in the hair is sometimes seen at a rather later date, and consists of thinning of the hair. The top of the head is most affected. Although true baldness is not present, the sparse condition of the hair may be rather striking, particularly when it is confined to the vertex.

Onychia is occasionally seen in infancy, especially in association with desquamative rashes. The nails become separated and blackened, and ultimately fall off. Should the child live, under mercurial treatment new and healthy nails grow.



FIG. 39.—"Syphilitic Wig"
INFANT, ABOUT 2 WEEKS, WITH DISSEMINATED

SYPHILIS OF THE BONES AND JOINTS.

Epiphysitis.—The commonest sign for syphilitic epiphysitis to appear is about the sixth week. It very rarely occurs after the third month.

The earliest symptom is pain, which causes the affected limb to be held motionless (syphilitic pseudoparalysis). At first no swelling is noticeable, but later thickening in the region of the epiphysal line becomes palpable and even visible (fig. 10). The infant screams on being handled. Crepitis may be obtainable from the separated



Fig. 10.—SYPHILITIC EPIPHYSITIS.—SWELLING ABOVE KNEE JOINT AND TENDerness.

epiphyses. The arms are more commonly affected than the legs. The lesions may or may not be symmetrically placed. Edema of the hands and feet occasionally develops with syphilitic epiphysitis.

The diagnosis is not difficult. If swelling is present, the case may be mistaken for infantile scurvy; if absent, acute polyomyelitis may be suspected. In both cases, the age-incidence of the disease should make the diagnosis clear. Scurvy is unknown before the fifth month of life, nor is the swelling of scurvy localized to the epiphysal line as it is in syphilitic epiphysitis. Other signs of inherited syphilis are usually observable to be found.

With mercurial treatment, preferably by inunction, the condition of the limbs improves rapidly and without leaving any permanent damage. Scott's dressing should be applied to the limbs.

Dactylitis.—Dactylitis is less common than the foregoing affection. It occurs in children under two years of age, and affects the fingers more often than the toes. It is said to be very symmetrical, but to this there are certainly many exceptions. It usually affects the proximal phalanges of several fingers, rarely the metacarpal bones. The disease develops as a fusiform swelling of the phalanx, often appearing to start from one of the epiphyseal regions. In the absence of treatment, the swelling increases, the skin becomes discoloured, and may break down and discharge necrotic material. The condition has been described as an epiphysitis, a periostitis, and as osteomyelitis. Its exact pathology is not yet known. Except by corroborative evidence of syphilis elsewhere, which is usually forthcoming, this form of dactylitis can hardly be distinguished from the tuberculous. With suitable treatment recovery soon takes place.

Periostitis.—A chronic periostitis is a late manifestation of inherited syphilis occurring during the later years of childhood until the age of puberty. The tibiae are the bones most commonly affected. In the middle-third of the shaft of the tibia there develops some swelling which is slightly tender. The anterior border of the tibia becomes rounded. Thus the bone appears to be curved with a forward convexity, and gives the condition the name of "*callos tibiae*." In some cases the bone becomes soft and actually bends. The skin over the swelling is sometimes reddened and pits on pressure, and ultimately necrotic material may be discharged through the skin. Other long bones may be affected.

In the long bones, and occasionally in the membrane bones of the skull, syphilis may produce small localized periosteal nodes.

Craniotabes.—The relationship of syphilis to craniotabes has been a matter of prolonged controversy. It is necessary to define first what is meant by craniotabes, for the term is used to denote two different conditions.

Craniotabes is used by some to describe a condition of diffuse yielding of the cranial bones, as a rule most marked at the angles and periphery of the frontal, parietal, and occipital bones. This is a condition which is normal during the first few weeks of life, and in some delicate infants may remain apparent until towards the end of the first year. This form of craniotabes, it seems clear, has no necessary relationship to syphilis.

On the other hand, by craniotabes may be meant a very different condition, in which there are small localized areas of parchment-like bone surrounded by well-developed bone. Such are most commonly found at the edges of the parietal and occipital bones. The term *craniotabes* is better limited to this change. It is due to absorption of bone, while the former condition is due merely to delayed bone-formation. This true craniotabes, however, can be the result of

rickets alone. It has been produced experimentally in rickety monkeys, and it disappears rapidly with antirachitic treatment. It is often associated with *Exiguus* nodulus and tetany, nervous symptoms of rickets. It seems very clearly proved, therefore, that craniotabes can be the result of rickets alone. As syphilis aggravates rickets and confuses to the worst type of this disease, it is only to be expected that in some cases of craniotabes, evidences of syphilis should be present. Syphilis without rickets is rare, and that it can produce craniotabes unaided seems doubtful.

Head-bossing (*Parrot's nodes*).—Here, again, there has been much discussion as to the relationship which syphilis bears to the condition in question. It is admitted that Parrot's nodes may be produced by rickets alone; and this some of the worst cases will be found when rickets has been aggravated by syphilis. The bossing of rickets is due to a vascular thickening of the diploe of the bone, the outer surface of which remains smooth. On the other hand, syphilis may undoubtedly produce increase in thickness of the bone, due to a deposit of rough, soft bone on its outer aspect, and with this type rickets has probably nothing to do. The conditions can be differentiated pathologically, but clinically this is impossible. In both forms the natiform or "bat-cross-bow" skull is produced, and there seems to be no true method of separating the ricketic form of the syphilitic cases. It has been said that in the latter, the bossing is closer to the anterior fontanelle, and that the edges of the fontanelle are thickened in rickets and thinned in syphilis.

Joint Affections.—Two forms of syphilitic joint affection occur, both in older children. The first is that of symmetrical synovitis of the knees, a chronic hyarthrosis, often associated with intersternal gonitis. It is generally painless. As a rule, this is but little influenced by general or local mercurial treatment.

Secondly, a form of chronic arthritis closely resembling osteo-arthritis with osteophyte formation and nodes like those of Heberden, has been described by Dr. Still as due to inherited syphilis. It appears unimproved by antisyphilitic treatment.

SYPHILIS OF THE ALIMENTARY SYSTEM.

Wasting.—The syphilitic is usually, in the inviolable phase of hospital mothers, a "beautiful baby form." Wasting, however, in greater or lesser degree, develops as the child gets older. A useful distinction may be made between the two types of wasting that arise. In the first, during the early weeks the child does fairly well, but with the onset of the symptoms of the disease wasting commences. This type may not be very serious, and with mercurial treatment the child may come to low weight. The second is far more dangerous. In it

The infant, from within a few days of birth, loses weight rapidly, and this in the absence of any obvious gastro-intestinal disturbances. By the time syphilitic symptoms arise, the child is noticeably thin, and has the "little old man" appearance with the "calf as lost" but of the skin. Such cases, although there may perhaps be suspected, can hardly be recognized with certainty, unless there is known to be syphilis in the family. This type of case is less common than the former, but seldom reacts well to mercurial treatment by the time it is first recognized. The child generally dies when it is a few months of age. No definite syphilitic lesions are found in the intestinal tract.

Syphilitic Infantilism.—In syphilitic children, development is often much retarded. As they grow up their stature may be considerably below normal, and their appearance very much more juvenile than it should be. The changes of puberty are usually delayed.

Teeth.—The *anti-dentition* shows no characteristic changes. The eruption of the teeth, in the absence of associated rickets, is said by some to be premature. The enamel of the teeth is often very thin, and early decay is the rule in the first dentition of syphilitic children. On very rare occasions Hutchinsonian characteristics have been found in the milk-teeth.

The *permanent* teeth may show very important characteristics. Typical Hutchinsonian teeth show the following peculiarities: They are widely separated from each other; are abnormally small; are "peg-shaped," the base being narrower than the cutting edge; their cutting edge has its corners rounded off and shows a deep central notch. These changes are only found typically in the central upper incisors. Less definite peculiarities may be found in the lower incisors. The first molars are sometimes "dome-shaped," due to their inhibited growth and the rounding of their angles. Typical Hutchinsonian teeth are only present in a small proportion of cases.

Stomatitis.—Condylomata may be present in the mouth, appearing as thick, white plaques, which are raised above the level of the mucous membrane. Stomatitis may exist in syphilis, as in other children, but does not appear to have any characteristic form. In stomatitis, the white areas are not raised, and appear thin and superficial, and are thus distinguishable from condylomata.

Glossitis occurs in a small proportion of syphilitic infants. Usually there is a localized patch of thickening of the epithelium; occasionally ulceration occurs. Gums of the tongue is found rarely in other children.

Hepatic Cirrhosis.—The liver is very frequently affected in inherited syphilis, but only seldom gives rise to any clinical

symphtosis. The commonest change is that of a diffuse, fine, intercellular fibrosis. The capsule of the liver may be unaffected, or may become slightly adherent. Occasionally it is thickened, and firmly adherent to the liver. In some cases the liver is irregular in shape, apart from the presence of granules. Such a liver is shown in Fig. 41. Portions of the organ are contracted, as the result of the cirrhosis, while in other parts there appears to have been an attempt at compensatory hypertrophy of the liver tissue.

Associated with the cirrhotic changes may be granules, often miliary in infants, and larger in the case of older children. The liver is occasionally of a bright green colour in infantile cases.

Where clinical symptoms of cirrhosis of the liver are present, the child is brought to the doctor for enlargement of the abdomen. On



FIG. 41.—*Dissected Specimen.* Liver removed, *PERICAROTID FIBROSIS*, and *ADHESION OF HYPOGASTRIC*.

examination, there are found to be marked enlargement of the liver and of the spleen. The diagnosis of syphilitic cirrhosis of the liver can hardly be made with certainty unless other signs of the infection are present, or there is a very suggestive family history. Tuberculous peritonitis is the commonest cause of ascites in children, and this together with malignant disease of the liver or of the glands in the peral focus, are the conditions which very closely simulate syphilitic cirrhosis.

With treatment by mercury and iodide, recovery may occur in older children; but in infants the condition is usually fatal.

Jaundice.—Jaundice may be present at birth in syphilitic infants. Less often it appears a little later. In both cases it is usually fatal if due to this disease. In some instances it is due to syphilitic obstruction of the common bile-duct.

Jaundice may occur in the cases of syphilitic hepatitis with ascites, but it is not a prominent symptom.

Peritonitis.—With the exception of perihepatitis and perisplenitis, we know nothing definite of syphilitic peritonitis. It is possible that such a condition may account for some cases of fetal ascites. Dr. Still has recorded such a case, in which there was an adhesive peritonitis associated with an interlobular hepatic carcinoma.

SYPHILIS OF THE CARDIOVASCULAR SYSTEM.

It has been abundantly shown that inherited syphilis has no direct influence in the production of congenital heart-disease. That it may predispose to it by lessening the reproductive power of the mother, is possible but unproved.

Intercardial myocarditis and a gummatous deposit in the wall of the heart have both been described in syphilitic infants.

Arterial changes are of more importance. Endarteritis of the cerebral vessels may be responsible for some of the abnormalities of the brain found occasionally in infected infants. That it may cause a hemiplegia from thrombosis is mentioned under the effects of syphilis on the nervous system.

It is very rare to find appreciable thickening of the nasal arteries during childhood. In some instances the patient is a syphilitic, and possibly there is an early condition of atheroma, due to endarteritis of the *nasa nasorum*, such as Dr. Mott describes in the acquired syphilis of adults.

SYPHILIS OF THE GENITO-URINARY SYSTEM.

Acute Nephritis.—This is not a common manifestation of inherited syphilis but is now a well-known occurrence. The symptoms commence between the sixth week and third month, but may appear at any time in infancy or childhood. There are, however, so few causes of acute nephritis at the period of life mentioned, that syphilis should be suggested in cases of that age. At the sixth month, herniations are usually due to scurvy. The nephritis of syphilis may be universal, but in some cases has been chiefly interstitial in type. The symptoms and signs do not differ from those of acute nephritis at other ages. Care must, however, be taken, lest the edema so commonly associated with diarrhoea be mistaken for this condition. Under mercurial treatment the symptoms quickly clear up.

Chronic Interstitial Nephritis is very rarely found in children. Even what has been said on the subject of the interstitial changes in acute syphilitic nephritis, it will be seen that there is some probability that syphilis may be a cause of the chronic disease.

Hæmoglobinuria.—Postnatal hæmoglobinuria is a very uncommon condition in children, but has been noted many times in connection with inherited syphilis. De Still states that it usually, if not always, indicates parental syphilis.

Orchitis.—This occurs within the first five months of life, and may affect one, but more often both, testes. No hydrocele is usually present. The testis becomes large, hard and abnormally insensitive. The condition is of no little importance, for two reasons. It may be of considerable diagnostic value, as it cannot well be mistaken for any other disease at the age of five months. Tuberculosis is uncommon at that age, and affects the epididymis more than the testis. With syphilis the former organ is rarely affected. Secondly, it is of importance because unless treatment is undertaken, the glands will later be functionless. With mercurial treatment, many but not all cases of orchitis clear up.

SYPHILIS OF THE SPLEEN, BLOOD, AND GLANDS.

Splenic Enlargement.—In about half the cases of inherited syphilis the spleen is easily palpable. It often projects an inch or so below the costal margin, but is rarely enlarged to a greater degree than this. The enlargement is uniform, and the organ feels hard. At autopsy it appears indurated when cut with a knife, and shows microscopically an excess of connective tissue. There may be areas of thickening of the capsule. Enlargement of the liver may be associated with splenic enlargement. Gaminiats of the spleen are extremely rare. The enlargement of the organ cannot be distinguished from that of rickets or tuberculous, apart from other evidences of syphilis, nor is absence of splenic enlargement a sign of much value against a diagnosis of syphilis.

Anæmia.—Anæmia is present during the mercuric stage of the first few months in inherited syphilis. The infant may show the peculiar brown or clay or *fox tint* at that time. But at a later age, usually during the second year of life, syphilis produces a more interesting form of anæmia, being one cause of "anæmia with enlarged spleen" which is so common in children at this age. The child is well nourished, but very pale, and perhaps rather yellow, but not of the same tint as the syphilitic infant. The blood shows the picture of secondary anæmia. The spleen is enlarged, and is palpable an inch or so below the costal margin. Definite evidence of rickets is usually present. The diagnosis rests between simple rickets, tuberculous, syphilis, and Von Jaksch's anæmia. The question is one of great difficulty, inasmuch as many do not admit that there is such a disease as Von Jaksch's anæmia, while it seems clear that both rickets and syphilis predispose towards that disease, should it occur. The diagnosis of

sypilitic *arthritis* is only to be made by signs of this infection elsewhere in the body, by a history pointing towards this disease, or by the serum test.

Adenitis.—Syphilitic adenitis, so common in the acquired disease, is very rarely seen in the inherited infection. When it occurs it is usually a late manifestation. It is sometimes found at the same time as interstitial keratitis. Any of the glands in the body may be affected.

SYPHILIS OF THE NERVOUS SYSTEM AND SPECIAL SENSE ORGANS.

Convulsions are not uncommon in syphilitic infants, and may be the immediate cause of death. They are however rarely associated with any gross intracranial syphilitic lesion of prenatal origin, and are to be regarded as of the same nature as the convulsions which occur in any toxicemic infants. Epilepsy may develop in later life.

Congenital Idiocy of any type is not uncommonly found in infected families. In the great majority of cases the syphilis acts only as a predisposing cause by deteriorating the parental reproductive power, but occasionally gross syphilitic lesions of the brain are found in association with congenital idiocy. Microcephaly with cerebral sclerosis, hydrocephalic meningitis, and endarteritis have been noted.

Juvenile General Paralysis.—This is the commonest form of mental deterioration associated with gross anatomical changes from inherited syphilis. It is an acquired and progressive condition. Starting, during the latter half of childhood (fifth to fifteenth years), the disease runs a course of three or four years' duration. Occasionally, as in adults, it is much more acute, and causes death within a few weeks of its onset. The early symptoms are those of headache, convulsions (usually mild in type), and mental deterioration. Transient hemiplegias may occur. Grandiose hallucinations, such as are found in the classical type of general paralysis in adults, are absent, but delusions may be present. The limbs at first are tremulous, and show the reflex changes of spasticity, the condition being one of acquired cerebral diplegia. In about one-quarter of the cases the knee-jerks are lost. Speech is slurring and indistinct. The pupils are often unequal, and of the Argyll-Robertson type. Towards the close of the case the mental deterioration passes into complete dementia, and contractions developing, the child becomes bedridden.

In other cases the symptoms are little more than those of progressive mental deterioration with signs of inherited syphilis.

The mental aspect of the disease differs in no way from that of general paralysis of the insane in adults. The condition is one of chronic meningo-encephalitis. The meninges are thickened and

adherent to the calvaria and brain. The frontal convolutions are atrophied, and the brain altogether is diminished in size. Some internal hydrocephaly is often present. The meninges are granular in appearance.

Apart from the rarity of the condition, the diagnosis presents no great difficulty. Progressive mental deterioration, with slight convulsions, spasticity, and signs of inherited syphilis, constitute the clinical picture of the disease. For the recognition of the syphilitic infection, the presence of Argyll-Robertson pupils and of choroido-retinitis are of the greatest importance, although other signs may also be present.

The prognosis is hopeless. The course of the disease is rather longer than in adults, and usually three or four years elapse before death occurs. Treatment is of no avail in preventing a fatal termination.

Juvenile Tabes Dorsalis (due to inherited syphilis) is very rarely seen under the age of twelve, but has been recorded in a boy eight years old. The symptoms are those of slight ataxia, deterioration of sight, and acquired incontinence of urine. The diagnosis is, as a rule, not suggested until an examination of the patient is made. Absence of the deep reflexes is then found, together with Argyll-Robertson pupils, optic atrophy and choroido-retinitis, may also be present. Later mental symptoms usually develop, and the case passes into one of the tabetic type of general paralysis.

Hemiplegia occasionally occurs in later childhood, from thrombosis of a cerebral vessel, due to syphilitic endarteritis. The paralysis may be preceded by warning attacks of paresthesia and headaches, and accompanied by loss of consciousness. The radial arteries in these cases may be palpably thickened. The diagnosis is made by the signs of inherited syphilis, particularly in the eye-ground. The prognosis, as regards the actual hemiplegia, is good; but the ultimate condition of the patient is likely to be one of general paralysis.

A **transient hemiplegia** may occur during the course of juvenile general paralysis, or in adult cases.

Meningitis.—The form of syphilitic meningitis which is most common in the inherited disease is that already described under general paralysis. Of localized syphilitic pachymeningitis, so common in the acquired infection, little is known in inherited syphilis. It is possible that such may originate some cases of hydrocephalus and cerebral paralysis. In the former, the enlargement of the head usually dates from the first few months of life. Most cases of supposed syphilitic meningitis in infants are in reality instances of meningococcal infection.

Intracranial Gummæ.—For gummata to give rise to the symptoms

of intracranial tumour is most exceptional in inherited syphilis. Only a few cases have been reported.

Choroidoretinitis.—Although this may be present very shortly after birth, and possibly even at the time of birth, it is more commonly formed in the later than in the earlier years of childhood. It is of such frequent occurrence that it becomes of much diagnostic value. It may be present without any symptoms attributable to it being elicited, or it may cause partial loss of sight. Nystagmus may also be associated with choroidoretinitis, and this is the commonest cause of nystagmus in syphilitic children.

On ophthalmoscopic examination, dark patches of pigment of irregular shape may be found in the fundus, associated with white areas of atrophy. In less typical cases there are no patches of atrophy, but small dark dots of pigment peppered over the fundus, and often most marked at the periphery of the field. The first type is certainly syphilitic, the second is nearly always syphilitic in children. There may be opacities in the vitreous.

The diagnostic importance of choroidoretinitis is very great. It can exist without causing any appreciable disturbance in the vision, and it is particularly to be looked for in cases of disease of the nervous system due to inherited syphilis. Dr. Still states that it is present in 15 per cent of syphilitic infants under one year.

Iritis.—When iritis occurs, it is most commonly at the fifth or sixth month of life. According to Hutchinson, it is more frequent in females than in males, and is almost always associated with some other symptoms of syphilis. It may occur in one or both eyes. The symptoms of iritis are not as a rule severe, but there is great danger of a resultant occlusion of the pupil. The disease is readily cured by mercurial treatment, but previous treatment does not seem capable of preventing it.

Later in life iritis may complicate interstitial keratitis.

Interstitial Keratitis.—This is one of the later manifestations of inherited syphilis. Beginning between the sixth and twelfth years, one eye is affected for several weeks before the other. In the earliest stages, the centre of the cornea becomes cloudy, and within this area of haze, as the disease progresses, there develop denser opacities in the form of small white spots which gradually coalesce. Together with these changes, abundant fine blood-vessels are seen running over the surface of the cornea. During the worst stages, the sight is almost entirely destroyed. The disease is one of long duration. In the most severe cases, the corneal opacity diminishes to a very considerable extent after many months, but in mild instances it rapidly disappears. Relapses occur in a small proportion of cases. The disease is one which reacts poorly to mercurial treatment.

Intestinal keratitis, as Hutchinson stated, is seldom seen without the characteristic deformity of the teeth.

Deafness.—This is one of the most serious symptoms of inherited syphilis. It is very rarely seen during childhood, and most commonly occurs at the periods of puberty or adolescence. The deafness comes on rapidly, unassociated with pain, otorrhea, or tinnitus, and in a few weeks hearing is completely and permanently lost. The cause of the deafness is not accurately known, but is supposed to be an inflammation of the internal ear.

During infancy, otitis media may develop as the result of the nasal obstruction associated with snuffles.

DIAGNOSIS, PROGNOSIS, AND TREATMENT OF INHERITED SYPHILIS.

Diagnosis.—The diagnostic points of the various manifestations of the disease have been dealt with in the foregoing pages. Attention will here be paid to three points only, which may be of great value in the diagnosis of any of the forms of inherited syphilis.

Firstly, chondrodermatitis is of particular value in the diagnosis of syphilis of the nervous system. Although it may be present with any of the manifestations of the disease, it is most frequently found with the nervous lesions. It may occur in early life, but becomes more common in later childhood. It is to be remembered that it may exist without causing any symptoms and consequently its presence cannot be excluded without ophthalmoscopic examination.

Secondly, syphilitic orchitis is of particular help in cases of the disease in early infancy. At three months of age there is nothing with which the condition can be confused. Later, it is easily distinguished from a tubercular lesion, which usually affects the epididymis rather than the testis.

Thirdly, the serum test for inherited syphilis is of great value. On results of the test as simplified by Fleming (*Lancet*, May, 1904), the author has had considerable experience, and would regard a positive reaction as very strong proof of an infection by syphilis in a child, and a negative result as good evidence to the contrary. It is very difficult to cause this reaction to disappear by mercurial treatment in the inherited form of the disease. Such a result has, however, been recently recorded in one case following the use of "606."

Prognosis.—This has been sufficiently indicated in the preceding pages.

Treatment.—**Feeding.**—During infancy, the syphilitic patient suffers, as a rule, a most defective power of assimilation. Vomiting and diarrhea are very prone to occur, and wasting is a most prominent

symptom of the infection. Even in the absence of obvious gastrointestinal derangement, manifestations of the severest degree may be found. In short, the syphilitic infant is difficult to rear. For this reason it is of the greatest importance that the child should be given the benefit of its mother's milk. By Colles's law we know that a wet-nurse may become infected through suckling a syphilitic infant, so that such a procedure is absolutely unjustifiable. The question arises, is it always safe, as Colles's law states, for the mother to suckle her own child, even where she has shown no symptoms of syphilis herself. Exceptions to the law have been recorded on very rare occasions, and even if they should be admitted (which is a matter of great doubt), the risk to the mother must be so extremely slight that it may be entirely disregarded.

Wherever, then, it is by any means possible, breast-feeding by the mother should be insisted upon, as giving the child its best and perhaps its only chance of survival.

Drug Treatment.—Mercury is well tolerated by syphilitic infants. Solicitation from the use of the drug is extremely rare. It is held by some that the administration of mercury may have a deleterious effect upon the teeth, but this, which is at most doubtful, cannot be weighed against the general harmful effect of the disease imperfectly treated, and we cannot allow such a consideration to discountenance the value of treatment by mercury over a prolonged period.

The chief drawback to the use of mercury is that it may induce diarrhoea, and this is the possibility that has to be kept in view during the administration of the drug. The preparations of mercury which are of most use are grey powder, calomel, liq. hydrargyri perchloridi, and argenti hydrargyri.

For practical purposes treatment by means of inunction (arg. hydrarg.) is the most efficient, and it is probably never wise to rely upon the internal administration of mercury alone.

For internal administration, grey powder is the most useful, as it is the least likely to produce diarrhoea. To a small, wasted infant, in whom diarrhoea may be a real danger, it is wise not to give more than $\frac{1}{2}$ -gr. doses three times a day. Should the bowels become loose the mercurial may be combined with some aromatic powder of chalk (2 grs.) or a small dose of Dover's powder. In most cases mercurial inunction may well be employed in addition. In more robust infants and in older children, larger doses of grey powder may be given if necessary.

Calomel is said to act more rapidly than grey powder. It is, however, more likely to produce diarrhoea. It may be ordered in amounts up to $\frac{1}{2}$ gr. two or three times per diem.

A fluid preparation may be of use in cases where it is desirable to give some other mixture, or where powders are vomited. Here liq. hydrarg. perchlor. may be given in doses of 3 to 5 drops to a small infant. It may usually be added to a castor-oil mixture, with which very small doses of tinct. opii or tinct. camph. co. may be combined if necessary.

Administration of mercury by inunction is the most useful method by which the patient can be put rapidly under the influence of the drug. For this use, hydargyrum may be used, a small quantity about the size of a pea being rubbed gently into the skin once, or if necessary twice, in the day. The ointment may set up some dermatitis, and for this reason the inunction should be made alternately in different regions of the body. The axilla, the four quadrants of the abdominal wall and inner aspects of the thighs may be used as the sites for inunction. The ointment is smeared gently rubbed into the skin by warm fingers for a few minutes, and then covered with a flannel bandage for twelve hours. The skin, at the end of this time, is carefully washed and powdered. The method may be used alone or in combination with internal administration of the drug.

As to the duration of the treatment by mercury, the drug should be taken for at least one year after the subsidence of active symptoms. Probably even a longer period of treatment is desirable. I have not yet seen treatment by mercury cause a positive syphilis reaction to disappear in inherited syphilis.

Potassium iodide is well taken by children, and where indicated can be given in large doses.

General hygienic treatment, especially by means of fresh air and good food, is of the utmost importance in the treatment of inherited syphilis.

VII—THE MENINGOCOCCAL INFECTION.

It is now generally held that there is no essential difference between the diplococcus of posterior basilar meningitis (described by Dr. Still) and the diplococcus intracapsular meningitidis of Weichselbaum, the cause of epidemic cerebrospinal meningitis. Such slight differences as exist are thought to represent, as Dr. Still originally suggested, "rather a modification of characteristics than a distinction in kind" and both types are now included under the term meningococcus.

Clinically we have, as has been shown by Dr. Langmead (*Practitioner*, April, 1907), a distinct link between the infantile and adult types of the disease in the manifestation of the infection as seen in older children. Pathologically, too, the differences are extremely slight. So that, considering all points, the epidemic disease is considered by most authorities to be a much more virulent infection due to the same organism as causes the sporadic or post-larvæ cases.

More recently, however, it has been found that there are certain differences in the clumping reactions and opsonic indices of the two conditions. The serum from an epidemic case may cause clumping of the epidemic organism, but does not affect that of posterior basilar meningitis, and, conversely, the organism of the epidemic disease is

not clumped by the serum from a case of post-basic meningitis. The inoculation also of a vaccine from one type of disease causes no variation in the agglutination index to the other organisms. It must be remembered, however, that the clumping reactions are not definite agglutination tests, such as are done in the Widal's reaction in typhoid infection, but that very slight dilutions are used.

How far these differences prove that we are really dealing with two infections and not one, is not at the present time settled. There appears no doubt that the post-basic cases in infants, and the epidemic cases in adults, are exactly the same infection; but it is possible, although at present the balance of evidence is rather against such a view, that the epidemic infection is both children and adults is another, and not only a more virulent type of the same disease. In this connection it is interesting to note that Dr. Langmead states that the ordinary cases of post-basic meningitis in infants, often form in reality very small epidemics.

Most hold that there is but one disease, and at the present time both forms of meningococcal meningitis are notifiable. This view is adopted here; but until it has proved to be correct, the clinical conditions are best described under their old names, as posterior basic, and epidemic cerebrospinal meningitis. Perhaps at some future time we shall be able to regard them as cases of endemic (sporadic) and epidemic, or of simple and malignant, meningococcal meningitis.

POSTERIOR BASIC MENINGITIS.

Etiology.—The age-incidence shows that there is a greater tendency to this disease during the first year of life than at any other time, and that the majority of the cases occur between the sixth and ninth months. It is a disease of the cold months of the year, especially from January to March. Apart from defective hygienic surroundings, we know of no other predisposing factors. The second cause of the disease is the organism described by Dr. Still, a Gram-negative, intracellular diplococcus.

Symptomatology.—Not uncommonly severe catarrhal signs, running from the nose, bronchitis, and occasionally broncho-pneumonia or diarrhoea, are observed for two or three days before the onset of the meningeal symptoms. Then rigidity is noticed, often ushered in by a convulsion in which the head may be retracted. At first there is considerable stiffness of the neck, but the retraction, which early is only slight, becomes more marked as the disease runs its course, and may become so extreme as to cause the occiput actually to touch the scapula. In addition there is some screaming, especially when handling the infant, but the "hydrocephalic cry," the shrill piercing yell which has been so much emphasized, is comparatively rare. The anterior fontanelle is bulging, and this points definitely to the presence of intracranial

disease. Usually some vomiting is present, and this tends to occur throughout the course of the disease and may be grouped into periods lasting three or four days. Constipation is commonly found, but not infrequently there are intermittent attacks of diarrhea which may be severe, as is shown by the sudden depression of the fontanelle. After the first few days the infant usually loses its power of swallowing and has to be fed by a nasal tube. The spas do not, as a rule, show any spitting until late in the disease, and when present this indicates extension forward of the meningitis to the anterior base of the brain. There remain these very important and characteristic signs to be



Fig. 42.—Infantile hydrocephalus. A case of Congenital Noncommunicating Hydrocephalus, when 2 months.

mentioned. Of these, the last is the loss of sight which occurs in the great majority of the cases, and is not associated with any changes in the fundi of the eyes, nor with any paralysis of the pupils. It is dependent upon the loss of function of the cells in the occipital regions of the brain, but it does not always mean that there is actual meningitis present over these areas. Later, should hydrocephalus develop, the pupils become dilated and paralyzed. The second characteristic sign is the so-called "comet stare," which is due to spasmodic retraction of the upper eyelids (Fig. 42). When marked, it is extremely suggestive of this disease. The third special sign is one of flex-

importance, and consists of perpetual shewing movements of the mouth and jaw.

As the disease progresses, the clinical picture becomes a very characteristic one: the child is unconscious, and lies with its head



FIG. 41.—POSTERIOR BASIC MENINGITIS: MORPHIC OPHIDIOFORMES, EQUILIBRY: MR. KRAMERSON.

thrown far back and limbs rigid; the legs are usually in a position of extreme extension, and the arms either extended or flexed: the fever is often very irregular in type: convulsions are common.



FIG. 42.—POSTERIOR BASIC MENINGITIS: MORPHIC OPHIDIOFORMES AND EQUILIBRY.

and there is great wasting (Figs. 43 and 44). It must, however, be mentioned that occasionally the disease runs a very much quieter course, in which the child appears only to be drowsy and partially unconscious, and the real nature of the condition passes unrecognized.

Towards the close of the active stage of the disease a purulent nasal discharge commonly develops. It is probably wise to treat this discharge as mildly infectious (p. 201).

A lumbar puncture reveals that the cerebrospinal fluid is under pressure, and is slightly turbid after the first day of the disease. On a microscopical examination, a large number of polymorphonuclear leucocytes are seen, many of which contain the diplococcal organisms which may be grown easily on various media. After two or three weeks, the fluid becomes clearer and contains fewer cells, most of which are lymphocytes, and a few swollen, vacuolated and dead diplococci. Occasionally, the exudate on the cord is so thick that no fluid can be obtained by lumbar puncture.

In older children the disease differs in some respects from that seen in infants, and more closely approximates to the sporadic form of cerebrospinal fever, as seen in adults. The head retraction and the cerebral stare are less marked, herpes is common, and optic neuritis is the rule. There is much pain in the head and occasional vomiting. Sometimes these cases show a peculiar temperature chart, in which pyrexial attacks lasting only a few hours occur regularly at intervals of about thirty-six hours. Their course is often one of several months, and as a rule the diagnosis is only made with certainty by the examination of the cerebrospinal fluid.

Morbid Anatomy.—The creamy purulent exudate is first seen on the under surface of the cerebellum round the medulla, and is then sharply confined to the area of the cisterna magna. Should the meningitis spread, it is found at the anterior base and spreading up the Sylvian fissures on to the cerebral hemispheres, which in very extreme cases may be covered. The spinal fluid of the lateral and fourth ventricles may be granular in appearance, and frequently in fatal cases there is pus present in the posterior horns of the lateral ventricles and in the fourth ventricle. The choroid plexuses may be shrunken and bound down to the floor of the lateral ventricles. Associated with these changes, it is the rule to find post mortem some dilatation of the ventricles of the brain, with flattening of the cerebral convolutions.

Most commonly death does not occur until the original purulent base focus is beginning to clear up, and only the remains of the purulent meningitis are found there (Fig. 45), together with adhesions which bind the medulla to the cerebellum, and are the cause of the secondary hydrocephalus which ensues. The spinal cord is always affected, and shows areas of purulent meningitis. These are better marked on the posterior than the anterior surface, and may be very small in extent, but occasionally there is a thick deposit all over the cord. The grey matter is, as a rule, slightly injected.

Microskopically, comparatively little change is found in the cells of the brain or cord.

Diagnosis.—It is rarely difficult to be sure that there is meningitis present; but occasionally, the disease starting with bronchopneumonia, its meningeal nature may be overlooked. The condition of the anterior fontanelle is of great importance, although there may be meningitis without any bulging of the fontanelle, yet this sign in an infant with an acute illness, in the absence of sweating or convulsions, almost certainly indicates meningitis. Retropharyngeal abscess causes head-retraction, but the other signs of meningitis are absent. The type of meningitis present is as a rule easily distinguishable by the age of the patient, the head-retraction, blindness, staring, and the movements of the jaws, with the absence of optic neuritis (see Table, p. 127). In a few cases only, the clinical evidence is indefinite, and here the examination of the cerebrospinal fluid will decide the nature of the case.

Very rarely severe cases of cerebral sclerosis in infants may show head-retraction, blindness and opisthotonos. They are distinguished by the absence of bulging of the fontanelle and the normal state of the cerebrospinal fluid. Tuberculous meningitis with an atypical posterior basal distribution (p. 125), and influenza meningitis (p. 143), may exactly simulate the ordinary meningococcal post-toxic meningitis. These conditions, both of which are very rare, can only be recognized by an examination of the cerebrospinal fluid.



FIG. 42.—Posterior basilar meningitis: removal of pit in typical position, with extension posteriorly to anterior base.

Course and Prognosis.—Of cases of this disease, about 50 per cent die and about 15 per cent recover completely, the rest being permanently injured by hydrocephalus, mental deficiency or deafness.

In the cases that are going to recover, after a period of fever lasting for three or four weeks, the symptoms gradually abate, and the child regains its sight and its ability to swallow; at the same time the rigidity passes off and there is an increase in weight.

Of the fatal cases, a few die during the first week from toxæmia or convulsions; but by far the greater number die towards the end of the febrile period, as the result of increasing hydrocephalus, associated, it may be, with extension of the meningitis over the brain or into the ventricles. Some, however, die of hydrocephalus, where there has been an active inflammation of the meninges at the posterior base only.

Death may not occur from this cause in several months or even years. The actual termination of cases of this disease is not uncommonly sudden, and is probably due to the pressure of the fluid on the vital centres in the floor of the fourth ventricle.

Should the patient survive, the prognosis as regards sight is important. In most infantile cases, sight is restored, but in older children there is a great danger of post-herpetic optic atrophy.

In any particular case, the prognosis is one of great uncertainty. The presence of a squint, of many convulsions, of the signs of hydrocephalus, are all of bad import, as showing extension of the meningitis; but as has been mentioned, fatal hydrocephalus may result from a lesion which is strictly localized to the posterior base, leading to adhesions being formed there.

Treatment.—As at present practised, this is very unsatisfactory. The child must be carefully nursed, and usually for a long period tube-feeding has to be undertaken. If there is much restlessness or screaming, bromides and chloral may be given. Lumbar puncture, although theoretically it should do good, is of very little benefit except in relieving the convulsions that may occur: the rigidity and opisthotonos are only temporarily improved, and vomiting is very little influenced by this measure.

Mercury and iodide administered internally have been used as routine measures in the treatment of this disease, but they do not appear to exercise any definitely beneficial influence upon its course.

There are various sera prepared for use in these cases, of which Reppel's and that prepared and sold by the Lister Institute are probably the most valuable. Occasionally, the intraspinal administration of the serum is followed immediately by very great improvement and, indeed, a rapid cure of the disease may date from its use. More often, however, it seems entirely without effect. It is better given intraspinally than subcutaneously as directed, its injection being preceded by the withdrawal of some of the cerebrospinal fluid. Of the value of the other sera recommended for use in posterior base meningitis, it is as yet too early to speak.

On theoretical grounds, it would appear that vaccine treatment might very properly be undertaken in posterior base meningitis. The disease is one which runs a course of several weeks, and in which it is easy to isolate in pure culture the causative organism. Further, it is an infection in which medical measures are practically useless, and one in which it only a little benefit can be obtained by treatment, there may result ultimately a complete instead of an incomplete recovery. While by means of vaccine-therapy we can only attack such organisms as can be reached by the blood-stream through the meningeal vessels (those in the cerebrospinal fluid being unapproachable by this method), nevertheless I am satisfied that in several cases I have seen beneficial results from this form of treatment. It should

be begun at the earliest possible moment—for clearly nothing can be of benefit when adhesions are formed—and should be combined with intraspinal injections of antismeningococcal serum. The initial dose of vaccine for an infant is from 2½ to 5 million coccæ, and at present the injections are best guided by estimations of the opsonic index.

EPIDEMIC CEREBROSPINAL FEVER.

It is not here necessary to give a detailed account of this type of the meningococcal infection, for it does not differ from the disease of adults. The epidemics occur in the winter months. The disease is not so much limited to infants as is the sporadic infection, but it is more common in children under ten years of age than in other subjects.

With a sudden and overpowering onset, the disease usually runs a very virulent course, with a high fever; and death may occur within a few hours of the initial symptoms, but more usually within the first week of the illness. The child presents more the picture of a pneumococcal meningitis than of a posterior basic inflammation, as is to be expected when it is remembered that in these cases the vertex of the brain very quickly becomes involved. Convulsions, rigidity, slight head-rotation, tremor, with severe collapse and toxæmia are usually seen, and purpuric rashes are very commonly present (p. 203). Optic neuritis usually develops if the patient lives long enough.

On the other hand, during the prevalence of an epidemic, a few abortive cases may be seen in which the child suffers from a convulsion, followed by unconsciousness and rigidity. In the course of a few days the symptoms pass off entirely, and the child rapidly gets well. Here the diagnosis is only to be made with certainty by the examination of the cerebrospinal fluid.

Infectivity.—On this point there is much that is uncertain; but it appears clear that the patient is not personally infectious. It would seem that the disease is comparable to acute poliomyelitis, of which epidemics are not uncommon, where all the evidence points to the fact that the infected subject is quite harmless. At present, little beyond this fact is known, and it is probably safest to disinfect the patient's clothes, and the house in which the disease has occurred. The poorer classes are those most affected, but not necessarily those of the dirtiest surroundings. As the meningococcus has been reported as present in the nasal passages, antiseptic nasal douches are recommended as a prophylactic.

The Diagnosis can as a rule be made without difficulty during an epidemic; but the only conclusive proof is the recovery of the meningococcus from the cerebrospinal fluid.

The Prognosis in a case of any severity is extremely bad; but, as

has been mentioned, the slight "abortive" cases recover. With the use of Flexner's serum the death-rate of this disease has been much diminished.

Treatment.—For the pain and convulsive movements, chloral in full doses should be given, but morphia is better withheld unless absolutely necessary. Phenacetin may be of use. Hot baths form probably the most effectual sedative measure.

In the treatment of cerebrospinal meningitis we cannot, perhaps, hope for very good results from vaccine inoculations in a disease so virulent and rapid in its course. Flexner's serum, given in large doses by intraspinal injection, appears of undoubted benefit, and will almost certainly in the future be the line of treatment adopted. Other sera have been



FIG. 2.—INFANT, CEREBROSPINAL MENINGITIS. INTERNAL VIEW.

prepared, but do not seem to have any very marked action. Flexner's serum can be obtained from many hospitals, but is at present not yet on the market.

The subcutaneous injection of the patient's own cerebrospinal fluid may cause an alleviation of the symptoms. The organism does not grow in the subcutaneous tissues, consequently this form of treatment is analogous to that by a vaccine, except that an unknown number of organisms is given in this method. Now that vaccines can be so quickly prepared from the patient's organism, the chief advantage of the use of the cerebrospinal fluid subcutaneously hardly compensates for the possible danger of over-dosage. Injections of 5 cc. of the cerebrospinal fluid are recommended to be repeated at intervals of two or three days.

ARTHRITIS.

Arthritis is occasionally met with in meningococcal meningitis, of either type, and may possibly arise as a primary condition. It is usually no more than a slight swelling of the affected joint, but occasionally the arthritis forms the chief symptom of the infection.

and the meningial disease may not develop until later. As a rule, beyond putting the limb at rest, no treatment is necessary, the swelling quickly subsiding and no injury to the joint remaining.

SKIN MANIFESTATIONS.

Purpura is seen in from 25 to 30 per cent of the cases in an epidemic, but probably if only children were considered, the proportion would be larger. The rash usually consists of petechial hemorrhages scattered over the trunk and limbs. Larger purpuric patches may be seen, as shown in Fig. 46, and, rarely, extensive subcutaneous hemorrhages develop.

Herpes is found in the epidemic cases and occasionally in the epidemic of older children. It is very rare in the ordinary infantile posterior form cases.

VIII—TYPHOID AND ALLIED INFECTIONS.

TYPHOID FEVER.

Little need be said on the manifestations of typhoid fever in children. Excepting infants, as a general rule the infection is less severe and the prognosis better than in adults. It is under the heading of the diagnosis of typhoid that the chief points of interest are found in connection with the disease in children.

Infants are not exempt from the liability to typhoid, but the great majority of the cases in children occur after the age of four. The onset is insidious as a rule, with headache and constipation; or, less commonly, diarrhea. Two points may be mentioned: that vomiting at the start of the disease is not uncommon in children, and that some exceptional cases, as in adults, start with great suddenness with a convulsion, rigidity and unconsciousness. With the rash, enlarged spleen, bronchitis, and the abdominal symptoms there is no need to dwell; they are subject to the same variations as in adults. The course of the temperature requires a word of mention. It is not uncommon in a mild attack of typhoid in a child, for the fever to disappear within ten days or a fortnight, and in some cases the temperature may be remittent, and profuse sweating may occur. It is, however, more usual for the chart to appear very similar to that of an adult case, showing the same length and type of fever. Relapses are quite common in children. In severe cases, the fever may be protracted just as in adults, and the cerebral signs marked. Widal's reaction behaves exactly as in the typhoid of older subjects.

Prognosis.—In infants the outlook is bad, but in older children the prognosis is more favourable than in adults. In them it is not often fatal. A few, however, die as the result of intestinal perforation, and still fewer of heart failure. In any particular case the prognosis will be framed on lines similar to those applicable to adults.

Diagnosis.—When we consider the differential diagnosis of typhoid fever we find that the difficulties are many, and are to some extent different from those which have to be solved in dealing with adults. It is perhaps useful to consider three groups of cases:—(1) Where the child is apparently only slightly ill; (2) where severely ill; and (3) where severely ill with cerebral symptoms. It may be said at the outset, that a large number of cases where enteric fever is suspected prove themselves later to be of some other nature.

1. Where the child is only slightly ill, with a moderate temperature (102 F.), turned tongue, constipation, and showing a rather early complexion with flushed cheeks, we have to consider whether it is a case of simple constipation or an early stage of enteric fever. When we remember how ill a child may be from constipation alone, and how slight the symptoms of typhoid may be, it is no wonder that at first it may be impossible to distinguish the two states. The administration of an enema, however, together with some small repeated doses of calomel, soon settles the question. In other cases, the examination of the urine reveals pus to be present, and it is well to remember that a case of *crisis* due to the *B. coli* may very closely resemble a mild typhoid fever in its early stages. Or again, where there is much wasting and the child looks ill, has a distended abdomen, but shows no very acute symptoms, a case of enteric fever may be easily mistaken for one of abdominal tuberculosis.

2. In the second group we may suppose the child to show a higher temperature, to look more poisoned and distinctly ill. Should any abdominal pain be present, the question of appendicitis may arise as in adults. Here we have additional difficulties, for vomiting at the onset of enteric fever is not uncommon in children, while diarrhoea is far more common in cases of appendicitis in young subjects than it is in old, so that the consideration of these two symptoms is not so helpful as in adults. Apart from the local abdominal condition, the blood examination is, of course, of the greatest service, a leucopenia being present in enteric fever and a leucocytosis in appendicitis. The apathetic condition of the typhoid patient and the localized character of the abdominal symptoms in appendicitis are suggestive points.

Cystitis and pyelocystitis may produce very severe symptoms, and may again cause difficulty in diagnosis in this group of cases. Here, too, abdominal pain may be present. An examination of the urine shows the true diagnosis; it is acid, but contains pus and the *B. coli*. Rigors and fainting attacks are characteristic of the severe cases of acute pyrexia. Osteomyelitis with pyæmia, or malignant endocarditis

may also simulate enteric fever. Uremia in children readily gives rise to confusion.

3. Where cerebral symptoms, such as unconsciousness, rigidity, with possibly a squint or delirium, are added to the high fever, we have a different group of possibilities to consider. If delirium is the most prominent symptom, pneumonia should be carefully excluded, for this is the commonest cause of such a condition. The respiration-rate is of chief importance, together with the physical signs in the lungs, the apex of which should be examined with special care. Again, pneumonia may simulate a case of typhoid, when there is no true delirium present, by its possible meningeal signs. It must be remembered that occasionally enteric fever starts quite suddenly, and in this way may resemble pneumonia. Where a typhoid case begins with pneumonia, the diagnosis will rarely be made before the second week of illness. Otitis media may be mistaken for typhoid, but usually resembles pneumonia more closely (p. 172).

In the group of cases we are considering, the greatest difficulty arises from general miliary tuberculosis, and, as in children this is a very common disease, it should be the first thing to be thought of when signs suggesting a very severe attack of typhoid are found. Both diseases may show a very similar mode of onset, and many of the signs and symptoms are common to both states. Thus the enlarged and over-hard spleen, bronchitic sounds in the chest, the diarrhoea, and even blood in the urine, may be present in either. The symptoms of vomiting, constipation, and diarrhoea; and the unconsciousness, slight head-retraction, general rigidity, with Kernig's sign and squint, are again common to both diseases, while slight ankle clonus and an indefinite extensor plantar response may be found in some cases of typhoid in young children with severe meningeal symptoms, and do not of necessity point to true meningitis. There are a few points which may be of some help. The cerebral condition may show some slight differences: for instance, the child with typhoid, although unconscious, may, when disturbed by an attempt to feed him, fight very strenuously against taking any nourishment; but with meningitis the patient is more deeply unconscious, and makes a much less purposeful resistance when attended to. The rash, if typical in its form and distribution, is most suggestive of typhoid, but it must be remembered that in cases of tuberculosis, a skin eruption of an indefinite acneiform character is very common. To be of real importance in the diagnosis, the rash must consist of the true rose-red spots. A retracted abdomen is characteristic of tuberculous meningitis, and a tumid one of typhoid, but to this rule there are exceptions.

The most important points remain; they are four in number. First, the Widal test, if positive, points definitely to the condition being one of typhoid fever; but occasionally, as is well known, in the severest forms of typhoid, so agglutinating substances are developed in the blood, and the test remains negative. Cultures

may be taken of the stools or urine. Second, optic neuritis, where definite points very strongly to meningitis. Third, choroidal tubercles indicate tuberculous meningitis without doubt. Fourth, the examination of the cerebrospinal fluid is final: in tuberculous meningitis there is excess of fluid containing many cells, mainly lymphocytes in type, and usually tubercle bacilli (p. 124); while in typhoid, although the fluid may be increased in amount from oedema, it contains practically no cells.

It may be of use to remember that a history of an illness with prolonged fever and unconsciousness, followed by recovery, means nearly always, in a child, typhoid fever; is an infant it usually means post-natal meningitis. This may be a guide if the child is seen for convulsions or for a relapse.

Lastly, in any case resembling typhoid, where there is a strong suspicion of some such condition being present, and the Widal reaction is negative, the patient's serum may be tested in the same way as the other organisms which are considered below.

Treatment.—This does not differ in kind from that adopted in adult patients.

ALLIED CONDITIONS.

The various strains of the paratyphoid, Gaertner, paratyphoid and colic bacilli may be the cause of an illness running a course resembling that of typhoid, usually of a mild type. Two cases of Gaertner infection occurring in infants have been reported by Dr. F. E. Batten, in one of which small follicular ulcers were scattered throughout the small intestine, with the diarrhoea dysenteriae. These conditions are at present only recognizable by agglutination tests with the various organisms.

IX. THE GONOCOCCAL INFECTION.

With the exception of *ophthalmia neonatorum*, this is a comparatively rare infection in children.

Ophthalmia Neonatorum need only be mentioned to emphasize its frequency and its seriousness. A great majority of the children in the institutions for the blind are there as the result of this disease. The importance of routine prophylactic measures thus becomes obvious.

Arthritis is occasionally seen complicating the conjunctivitis in infants, and in older children may arise from vaginitis.

Vulvovaginitis is not in all cases due to the gonococcus, but may be streptococcal in origin and brought about by lack of cleanliness. The more severe cases are, however, usually gonococcal, and generally originate from the use of infected towels. Very rarely, extension may occur to the pelvic organs and peritonitis. The treatment is of importance in institutions, for the disease tends to spread amongst female children. Both types of case are dangerous, but particularly the gonococcal; these, where possible, should not be admitted or retained in a children's ward. Diapers should be worn, so that the child may not infect her eyes, and if necessary the arms should be kept straightened by means of a small light splint, so that the face cannot be touched. Frequent irrigation should be ordered; solutions of zinc chloride (a grain to the ounce) or potassium permanganate (one part in ten thousand) are perhaps the most useful. Antiseptic dressings should be applied. The disease is difficult to cure, and recrudescences are common. Epidemics amongst the children in a ward are very hard to stamp out. In the streptococcal cases, frequent bathing with boracic lotion, and the application of some mild antiseptic ointment, are usually sufficient to cure the disease. The presence of thread-worms may prevent the subsidence of vulvitis.

X.—THE INFLUENZAL INFECTION.

Only a short account of influenza need be given, since the infection as it occurs in children is broadly similar to that seen in adults.

Etiology.—The disease may occur at any age; even children in the breast may be infected from the mother. Children are most commonly infected during the epidemics which so frequently arise in the months of winter and spring. The incubation period of the disease is short, probably from one to seven days.

Complications due to the pneumococcus play a large part in the symptomatology of the disease. Those due to the *M. catarrhalis* are less common.

Symptomatology.—In most instances the symptoms agree with those seen in older patients. The coryza, pains in the head and body, fever, and severe prostration are characteristically present, and are followed by the same prolonged debility as in adults. The peculiarities at any particular epidemic may be found in the young subjects as in the older.

In infants, although mild attacks may be seen, the infection is not seldom of an intensely violent septicæmic type. Within a few hours the infant is unconscious, severely collapsed, cyanosed, dyspnoic,

and death occurs in two or three days. In a few cases of this group a suppurative meningitis, due to Pfeiffer's bacillus, is the cause of death.

In older children these septicaemic cases may also occur, and give rise to symptoms exactly simulating those of tuberculous meningitis. Indeed, such a diagnosis can sometimes only be excluded by the absence of the typical changes in the cerebrospinal fluid. Influenza, then, must be remembered as a cause of meningitis. As a rule, such cases as these recover, often to the surprise of those in attendance.

Symptoms from the Respiratory Tract are particularly common in children. Laryngitis, tonsillitis, tracheitis, bronchitis and pleurisy are frequently present. Pneumonia, associated with influenza, is seen in three groups of cases. In one, a pneumococcal pneumonia is induced in with influenza. The patient is at first severely prostrated and, if he lives, tends to improve as the pneumonia runs its course. The left ventricle of the heart is not uncommonly dilated in this condition. In the second group, during the febrile stage of the influenza infection, rather indefinite patches of consolidation are found to move about from one part of the lungs to another. As a rule, the pneumonic signs do not develop beyond the stage of slight dullness and crepitations. The third group consists of a peculiar form of broncho-pneumonia resembling somewhat that of the second group, but protracted over a period of many weeks. In such cases, which are usually regarded as influenzal, the pneumonic process creeps about the lungs, and as each transient consolidation occurs there is a fresh rise of temperature for two or three days. In other cases, the fever is more regularly intermittent in type. There is no little danger of regarding such cases as tuberculous; recovery is, however, usually complete.

Rashes are occasionally seen. They may resemble those of measles or scarlatina, but are more irregular in their distribution.

Dilatation of the Heart has already been mentioned as occurring during the acute stage of the infection. It is due to influenzal myocarditis. Cardiac sequelae are amongst the most serious results of influenza.

Influenzal Meningitis, due to Pfeiffer's *Bacillus influenzae*, is a rare condition which is more common in infants than in older children. The symptoms strongly resemble those of a meningococcal meningitis, and the nature of the infection can only be recognized by a bacteriological examination of the cerebrospinal fluid. The suppurative exudate is found on the vertex and at the base of the brain, and may extend into the ventricles or down the spinal cord. In older children recovery occasionally occurs, but in infants death is almost invariably caused by this condition. Dr. F. E. Bates, however, has reported a cured case in a child of fourteen months of age who was treated for three weeks by 10 grains of acetophenol daily.

Sequelae.—Post-influenzal debility and anæmia may persist for several months after influenza, and may predispose to tuberculosis.

Otitis media, catarrhal or suppurative, and enlargement of the cervical glands, are not uncommon during or following the infection. Bronchitis and tracheitis may remain troublesome for many weeks. The nervous sequelæ are not common in children. Mental depression is only transient as a rule; peripheral neuritis is very rare.

Cardiac Sequelæ.—Which owe their origin to infectious myocarditis, are not uncommon and may be very serious. A marked irregularity of rhythm, which may persist for many weeks, is a frequent result of infection. This is usually associated with some dilatation of the heart, and occasionally with the development of a systolic murmur. The majority of these cases get perfectly well; but we have no certain knowledge that such cardiac dilatation invariably disappears. Sudden death in a child who is thought to be well, but who has recently had influenza, is not a very rare event. It usually follows a bout of hard exercise. Infectious myocarditis is, with rheumatic and diphtheritic myocarditis, one of the causes of a sudden fatal syncope in a child.

Prognosis.—Except during infancy, the prognosis is better in children than in adults. Severe attacks in infants are usually fatal. In older children the dangers are chiefly due to the pulmonary complications, and pneumonia is the commonest cause of death during an attack of influenza. The importance of the cardiac sequelæ has already been urged.

As in adults, one attack predisposes to others.

Treatment.—This does not differ from that adopted in adult patients.

XI.—ACUTE POLIO-ENCEPHALOMYELITIS.

(*Acute Poli-encephalitis*; *Acute Polio-myelitis*.)

Introductory.—The disease acute poli-encephalomyelitis is due to an infection of the central nervous system which occurs more frequently in children than in adults, which is particularly prone to arise during the summer months of the year, and which is occasionally seen in well-marked epidemics. The chief symptoms originate from inflammatory processes which affect mainly the grey matter of the central nervous system. Most commonly these arise from involvement of the spinal cord (acute polio-myelitis), sometimes from the brain (acute poli-encephalitis), and sometimes from both brain and spinal cord together (acute poli-encephalomyelitis). The general constitutional symptoms are variable in their severity, and during epidemics of the disease cases can be recognised in which these symptoms are present without any paralytic signs due to destruction of nervous tissue.

It will be seen that we have here to deal with a disease much more widespread than that which was formerly known as infantile paralysis. There is ample proof that the lesions need not necessarily be confined to the spinal cord, and it is well to grasp the fact that cases in which the brain is affected are well known, and are in all respects similar to the spinal cases except as regards the localization of the lesions.

There is abundant evidence that the brain as well as the cord may be affected. Dr F. Buzzard, dealing with acute poliomyelitis in the Goulstonian lectures for 1907, pointed out that not only are the morbid changes not strictly confined to the grey matter of the cord, but that they usually extend far higher up the nervous system than is recognizable from the patient's symptoms during life. A fair number of cases of acute polio-encephalomyelitis have now been examined pathologically, and similar lesions have been found in the brain and spinal cord. In one which I have reported (*Mem.* 1907), there were symptoms of involvement of cerebrium, cerebellum and cord and, post mortem, morbid changes of a similar nature were found in all these parts of the central nervous system. Clinically, too, during epidemics, cases showing affection of the brain and cord are well known to occur; indeed, in very severe cases of the spinal form of the disease, it is not very rare to find evidence of slight damage to the brain. From these considerations, therefore, it is clearly proved that the brain is not immune to the infection, and that the damage done to it may be sufficiently serious to give rise to corresponding physical signs.

That the brain alone may be involved, is also clearly established. Only a few cases of acute polio-encephalitis have been examined pathologically; but clinical evidence points decisively to the existence of the condition. This is most clearly shown by the occurrence during epidemics of the spinal infection of cases of the cerebral type, without symptoms of any involvement of the cord. From this too, we see that the seasonal incidence of acute polio-encephalitis must be similar to that of acute poliomyelitis; and this rule held good in the cases of acute ataxia (cerebellar cases) which Dr F. E. Bates has reported, and in the series of cases of acute tremor (mid-brain cases) which I have brought forward. It has been pointed out that cases of infantile hemiplegia show no such definite seasonal incidence as does acute poliomyelitis; but it is to be remembered that infantile hemiplegia is a symptom and not a disease, and that no one would wish to suggest that all instances of the condition are due to acute polio-encephalitis.

In the following account of the infection, those points which are common to both the brain and cord cases will be taken together, after which acute polio-encephalitis and poliomyelitis will be considered separately.

Nomenclature.—The full name, acute polio-encephalomyelitis, is extremely cumbersome, but is not one of which it is necessary to

make frequent use, as most of the cases fit into one or other of its two subdivisions. It has, however, the advantage of describing the disease accurately as an inflammation of the central nervous system, in which the grey matter is chiefly affected. Inasmuch as it has been shown that in the spinal form of the disease the anterior horn cells are not the only parts damaged, the word "anterior" has been dropped from the former title, acute anterior poliomyelitis. To the disease as a whole, the term infantile paralysis is clearly inapplicable, for in some cases of acute poli-encephalomyelitis there is no paralysis, nor is the disease limited to infants.

Etiology.—Several clinical etiological factors are of interest. The type of child attacked by this infection is always the robust and healthy-looking child rather than the wasted and delicate subject. The age-incidence of the disease shows that it is most common between the sixth month and the end of the third year, with a maximum incidence in the second year of life. After the sixth year it is uncommon. The seasonal incidence is very striking. About three-quarters of the cases occur during the months of July, August, September, and October. In the sex-incidence there is no peculiarity, as the disease occurs in the first years of life, but after the age of ten, the males attacked far outnumber the females.

It has been stated that acute poli-encephalomyelitis frequently occurs during or immediately after an acute specific fever. Examining the admissions to the Hospital for Sick Children, Great Ormond Street, for acute poliomyelitis with its onset in 1900, I found only one case out of fifteen where there had been an acute exanthem within the three months preceding the paralysis. Dr. Leonard Parsons, investigating the same point at the same hospital for the cases occurring in 1903, found only one instance out of twenty-six cases within the three months limit. It would seem therefore, that such a sequence is far from common. In this connection it must be remembered that during an exanthem, an infective thrombosis may occur in the central nervous system, but this is probably a condition quite different from the infection which we are considering. On the other hand, the constitutional symptoms of acute poli-encephalomyelitis may be present before the development of those due to destruction of nervous tissue, and may be mistakenly regarded as those of influenza.

The disease is well known to occur occasionally as very definite epidemics. From this fact it is almost certain that the origin of the disease is bacterial. The disease does not appear to be infectious in the ordinary sense of the term. Probably epidemics arise from some common source of infection, rather than by spreading from child to child. The incubation period of the infection is as yet undetermined; it is probably within a week. Experimentally, in monkeys it averages ten days. One attack appears to confer complete and lifelong immunity against a second.

There is some evidence which suggests that the disease is due to a water-borne infection. The large epidemics usually follow the distribution of the water-courses, while small outbreaks have been thought to be due to bathing in infected water. It may be, however, that these points are to be explained respectively by the distribution and density of the population in epidemic areas and by the seasonal incidence of the disease.

Bacteriology.—The cerebrospinal fluid in this condition has shown no constant organisms, and very rarely reproduces the disease when injected into animals. During 1909 experimental workers have demonstrated several important facts bearing on the etiology of the infection. It has been shown that the spinal cord from a patient dying in the acute stage of the disease will reproduce the disease in monkeys if injected into the brain or peritoneal cavity, and that in this way the infection may be transmitted, not only from man to monkey, but from monkey to monkey. A glycerinated preparation of the spinal cord is similarly active and, a further point of interest, when the preparation of the spinal cord is passed through the finest form of filter, the filtrate will reproduce the disease. No organisms have, however, as yet been detected microscopically.

These facts seem to point to the conclusion that we have not yet at our disposal histological staining methods or microscopical magnifying power suitable for the recognition of the organism causing the disease.

Specificity.—It was formerly held that acute poliomyelitis-myelitis might be caused by various organisms, notably those of the acute specific fevers of childhood. It is, however, much more probable that we have in this disease a true specific infection. The age- and seasonal-incidences, the immunity afforded by one attack, its occurrence in epidemics, its obscure bacteriology, are all points in favour of its being itself a specific disease. Even should it follow an exanthem, this does not deprive its own specific character, for such a sequence among acute specific fevers is well recognised.

Morbid Anatomy.—The lesion may occur in any part of the central nervous system, and are usually more widespread than the clinical signs in the case would suggest. The grey matter is the part most affected, but the changes are not entirely confined to this, although it is rare for signs of the involvement of the white matter to be recognisable during life. There is no primary affection of the peripheral nerves.

The changes found are four in number: first, vascular changes consisting of hyperæmia, thromboses or minute haemorrhages; second, perivascular cell infiltration; third, cell infiltration into the grey matter; and lastly, necrosis of the tissue and destruction of the nerve cells of the grey matter.

These changes have been studied most in the spinal cord. It is to be remarked that the nerve-cells are destroyed only when they are involved in an area of cell infiltration, while those lying just outside such an area show practically no pathological change. From this it may be concluded that the causative agent of the disease has no special predilection for the nerve-cells, as has been often stated, but that the grey matter bears the brunt of the attack only because it is more vascular and of looser texture than the white matter. The nerve-cells appear to be mechanically destroyed by the inflammatory cell infiltration. The examination of the cord in a case of recent origin shows hyperaemia of the meninges over the focus of the disease. Later, considerable sclerosis may be present, and the affected anterior horn becomes shrunken.

There has been much discussion as to the nature of the primary changes in this disease. Formerly, it was thought that the important change in the spinal cord was that of the thrombosis of the blood-vessels, and that the cell infiltration and necrosis of tissue depended upon this factor. It is, however, now generally accepted that this view is incorrect, and it is held that the inflammatory cell infiltration is the primary change, the thrombosis being inconstant and, if present, of secondary importance.

Cerebrospinal Fluid.—Lumbar punctum gives no positive evidence of diagnostic value in this disease. There may be some slight excess of lymphocytes present in the cerebrospinal fluid, but there is no definite increase in the amount of albumin in the fluid. As has been already mentioned, no organism has been constantly present in it. The fluid taken from a patient in the early acute stage has reproduced the disease when injected into monkeys (Flexner), but in the great majority of cases it is experimentally inactive.

General Constitutional Symptoms.—Three types of the disease can be differentiated with reference to the constitutional symptoms of the infection. In the first and most common type, these symptoms are present, preceding and accompanying the local symptoms due to the destruction of nervous tissue; less frequently, no constitutional symptoms are present; while finally, during epidemics, there can be recognized instances in which these are the only symptoms, no local signs developing.

The constitutional symptoms consist of loss of appetite, with perhaps vomiting. Drowsiness and nocturnal delirium may develop, and convulsions may occur. Headache and pain in the back radiating down the limbs, are rarely complained of in children. Some rise of temperature is constantly present. It is interesting to note that the attack may be ushered in by a sore throat.

In exceptional cases the symptoms strongly resemble those of tuberculous meningitis, with drowsiness, cataplexic rigidity and

head-retraction. Such may be due to excess of cerebrospinal fluid (excess meningitis) and do not necessarily foreshadow the onset of cerebral or cerebellar lesions.

These general symptoms are in no way characteristic, and may exist for two or three days before the appearance of the local signs which enable a diagnosis of the nature of the infection to be made. As is to be expected, the general symptoms are more prone to be severe in acute poly-encephalitis than in acute poliomyelitis.

It is important to bear in mind that, whether constitutional symptoms are present or absent, the onset of the local signs is very sudden, and their development is complete, if not immediately, at all events within a very few hours. In the spinal cases, for instance, the paralysis, when it does appear, appears suddenly, and very quickly reaches its maximum. Anything like a puerus gradually becoming a paralytic, as in peripheral neuritis, is not seen in this disease.

ACUTE POLIO-ENCEPHALITIS.

The various forms of acute poly-encephalitis must be briefly mentioned, but it should be remembered that more than one part of the brain may be involved in the same case.

Polio-encephalitis Superior.—This name is now usually applied to the group of cases in which the cerebral cortex is affected, and this is divisible into *frontal*, *Rolandic* and *occipital* cases, according to the localization of the lesion.

Of these, the motor cortex is the part most commonly damaged; and it was in connection with such cases of cerebral hemiplegia that Strümpell first, in 1884, suggested the analogy between poly-encephalitis and poliomyelitis, so that this condition is sometimes termed Strümpell's paralysis. The face is usually only slightly affected, the chief paralysis falling on the arm.

Frontal and occipital cases are associated respectively with mental and moral deterioration, and blindness with normal eye-grounds and intact pupils.

Polio-encephalitis Inferior.—Here the nuclei of the various cranial nerves are affected. This is the commonest type of cerebral disease found associated with severe acute poliomyelitis.

Acute Ataxia (Cerebellar encephalitis).—This important condition was first described by Leyden in 1891, and recently many examples have been recorded by Dr. F. E. Bolton. The chief symptoms are those of marked ataxia and hypotonus of the limbs, to which are added, in some cases, dysmetria and scanning-speech. The ataxia is usually the most prominent, and may be the only, symptom. Scanning-speech is probably only present when the cerebellar lesion is bilateral, and consequently is evidence of a severe attack.

Only a very few cases have been examined pathologically, as there is but little danger to life connected with this type of the disease.

Experimentally, it has been found that a large amount of the cerebellum may be ablated without producing any permanent harm if the cerebrum is undamaged, and in these cases the improvement after the initial symptoms is usually very marked. In many there is complete recovery, while in all but a very few there is definite improvement. Slight cases recover in the course of a few weeks, but in severer ones improvement may proceed for months or even years. Evidence of severe cerebellar damage, as shown by the presence of scanning-speech or signs of cerebral involvement, make a guarded prognosis very necessary.

Acute Tremor (*Encephalitis of the cerebello-rubro-spinal system*).—

Another group of cases can be distinguished, of which the cardinal signs are those of tremor and hypertonia. These two symptoms have been shown by Dr. Gordon Holmes to be associated with destructive lesions in the red nucleus or in its connections with the cerebellum and spinal cord (the cerebello-rubro-spinal system).

The tremor is a slow rhythmic movement at the rate of about five oscillations per second. When well-marked it is constantly present, except during sleep. It may be more marked in the upper parts of the limbs than peripherally, and may be universally present throughout the body, giving an appearance very similar to one of shivering from cold. It is made worse by voluntary movements or by emotion. When less marked, the tremor is present only on voluntary movement. Combined with the tremor is hypertonia, so that movements of the affected part are slow, stiff, and awkward, and thus in the absence of true spasticity. The condition, therefore, strongly resembles that seen in paralysis agitans.

In addition, cerebellar symptoms proper, or evidence of involvement of the cranial nerve nuclei in the neighbourhood, may co-exist with the tremor and hypertonia.

The red nucleus and its connections form a system known as the cerebello-rubro-spinal system which consists of two parts. The cerebello-rubral portion connects the dentate body of one lateral lobe of the cerebellum to the red nucleus of the opposite side by means of the superior cerebellar peduncle, while the rubro-spinal portion (Moskowsky's bundle) connects the red nucleus with the opposite side of the spinal cord, probably terminating by synapsing round the anterior horn cells. In this system there are, then, two decussations, and both occur in the immediate neighbourhood of the red nucleus. It is held that a destructive lesion in any part of the system (dentate body, superior cerebellar peduncle, red nucleus or Moskowsky's bundle) will produce tremor. Should the lesion be in or close to the red nucleus, the tremor will be on the opposite side of the body, while if any other part of the system be affected, the tremor will be homolateral.

The prognosis in these cases is good. The tremor tends to disappear, and in all the examples which I recorded (Brous, 1921), ceased

to be of any severity. Where there is evidence of a large lesion, or where unconsciousness at the onset of the disease has been prolonged, there is a danger of mental deterioration resulting. In our such case, epilepsy developed and proved fatal.

Thalamic Encephalitis.—Acute polio-encephalitis has been reported as the cause of some cases of hemiplegia with clonus or athetotic movements in the paralyzed limbs. These have been regarded as due to cortical involvement, but in the light of Dr. Gordon Holmes's work it would seem more accurate to group them as examples of thalamic encephalitis.

Pontine Encephalitis.—This need hardly be separated from the cases already mentioned as polio-encephalitis inferior. The seventh and eighth nerves are affected. A few of this group show tremor and hyperreflexia from involvement of Monakow's bundles.

Fulminant Encephalitis.—This again is a subdivision of polio-encephalitis inferior. In it there is the sudden onset of bulbar palsy. These cases are not entirely to be fatal from asphyxia or from involvement of the vital centres in the medulla. Such changes have been found post mortem.

Fulminant Encephalitis of the Vital Centres.—It is possible that in some instances, the vital centres in the medulla are the only ones to be attacked, or at all events are the first to be destroyed. Such a condition possibly accounts for some of the cases which have been written of as "rapidly fatal constitutional diseases." Such cases may occur in small epidemics in institutions for children. After a few hours of slight illness, death occurs, usually with all the symptoms of acute asphyxia. The evidence of this group rests at present only on hypothetical grounds.

Diagnosis.—The diagnosis of acute polio-encephalitis in the absence of an associated spinal lesion can only be made by excluding other possibilities. Although the various etiological factors (p. 211) may be suggestive, there can be no positive evidence to confirm the diagnosis.

Tuberculous tumours and fibrinosis due to septalitic endarteritis are perhaps the most common sources of confusion. In the former, the type of child is usually very different from the infant subject of polio-encephalitis, and in addition, optic neuritis may be present. Moreover, it must be remembered, may take place in the case of such a tumour, just as in encephalitis. If fibrinosis due to septalitic endarteritis is suspected, valuable information may often be obtained from examination of the fundi of the eyes; for in cases of intra-cranial septalitis due to the infarcted form of the disease, chorioretinitis is frequently present. A careful examination should likewise be made for any other signs of the disease. In addition, there may be a history of warning attacks.

A few cases of tuberculous meningitis start with hemiplegic symptoms of sudden onset, as is mentioned in dealing with that

condition (p. 123). In the absence of any signs of tuberculosis in the other organs, or of recognizable constitutional degeneration, such cases can hardly be distinguished at first. Thrombosis may occur in marasmic infants; but these present no difficulty, as exophthalmos is more prone to attack the infant type of child. Infective thromboses occurring during, or shortly after, an acute specific fever, exactly simulate polio-encephalitis clinically. Such cases should not be included under the heading of polio-encephalitis.

The bulbar cases simulate the "bulbar crises" seen in post-diphtheritic paralysis, but in the latter other signs of peripheral neuritis are present (p. 241).

Treatment.—With the possible exception of the administration of potassium iodide in order to promote the absorption of the inflammatory products of the disease, we know of no measure of any avail in dealing with the cause of the symptoms, and the treatment must be undertaken on general lines.

The bulbar cases should be treated with full doses of atropine and strychnine.

ACUTE POLIOMYELITIS.

The etiological factors, morbid anatomy, and general constitutional symptoms of acute poliomyelitis have already been discussed (pp. 213-50, 214), and we have here to deal with the local or paralytic symptoms, the diagnosis, prognosis, and treatment of the condition.

Paralytic Symptoms.—The paralyses vary much in their distribution and extent, but are alike in that they are of sudden onset and very rapidly reach their maximal development. They are typical palsies of the acute lower neuron type, and are associated with flaccidity, rapid wasting, and the development of the reaction of degeneration.

The legs are affected six times as frequently as the arms. Where more than one limb is involved, the distribution of the paralysis is much more frequently paraplegic than hemiplegic in type.

In an ordinary case the affected limb is at first completely paralyzed, or perhaps movements of the toes or fingers are just possible. It is hyperæsthetic, movements or deep palpation of the limb producing pain. The explanation of this symptom is difficult; probably it is dependent upon the hyperæmia of the meninges over the spinal lesion. This hyperæsthesia usually passes off in a week or two, but may remain as long as six weeks. In a few cases areas of anaesthesia have been found in the paralyzed limb. The deep reflexes are abolished only at the muscle upon whose nerve they depend is paralyzed. Occasionally, an extensor plantar response has been obtained, indicating involvement of the whole matter of the cord. The limb is quite flaccid, and the utter loss of muscular tone in it is very characteristic. In the

course of a few days there is usually some improvement, especially in the peripheral muscles, while those which are severely paralyzed begin to show wasting and alterations in their electrical reactions. The muscles of the trunk may be involved, but it is rare for them to be attacked unless the limbs are also paralyzed. As a general rule



FIG. 21. CHILD PARALYZED WITH SEVERE ACUTE POLIOMYELITIS.

it may be stated that the muscles of the limbs are more commonly and more severely paralyzed than any other muscles of the body.

While the condition that has been sketched is the one most commonly seen in ordinary cases, there remain to be mentioned three other types of the spinal infection—namely, the very mild, the very severe and the relapsing cases.

(1) In the *very mild cases*, any group of muscles may be attacked, but most commonly it is the muscles about the hip or shoulder-joints that are paralyzed. In such cases during the acute stage there is some loss of power of movement at the joint, and often a good deal of

pain on examination, so that some inflammatory condition of the joint itself may be suspected. As the wasting develops and the hyperaesthesia passes off the true condition becomes plain. In the chronic stage, contracture of one part of one rectus abdominis muscle may be the only result of a larger infection.

The *very severe cases* are not extremely rare, and to one unfamiliar with the terrible amount of damage that the infection can produce, they may cause no little difficulty in diagnosis. It is well, therefore, to bear in mind the extent of the paralysis which may be caused by acute poliomyelitis. All four limbs, the muscles of the neck and trunk, the intercostals, diaphragm, muscles of the abdominal wall and the sphincters may be paralyzed. Of the respiratory muscles, the intercostals are more often affected than the diaphragm. The sphincter ani may be found to be quite lax, but should the child live, tends to recover its tone rapidly within a week or two. In these severe cases

it is not uncommon for some of the cranial nerve nuclei to be involved (acute poly-encephalitis inferior).

The *relapsing* cases are uncommon. When a relapse occurs, it is usually within a week of the initial attack. More than one relapse is very rare, but in the case already quoted as showing cerebellar, vestibular, and spinal symptoms, there were three relapses spread over a period of five weeks. Relapses, so far as I have noted, are not accompanied by any fresh constitutional symptoms.

Complications.—Where any of the muscles concerned with respiration remain paralyzed, there is a great liability to fatal bronchitis or pneumonia. It is said by some that the disease predisposes towards various nervous diseases, particularly disseminated sclerosis, in later life. In children, it is rare to find those who have had an attack of acute poliomyelitis coming under treatment for any other serious infection, a point which emphasizes the fact that it is the robust child who is subject to the disease.

Diagnosis.—With the exception of the very mild or very severe cases, there is seldom any difficulty in the diagnosis of acute poliomyelitis.

In a case where the paralysis is very limited, there may during the acute stage be much doubt as to the nature of the condition present, and it may be only on the development of the characteristic wasting that the matter can be settled. Particular difficulty may be met with where some of the muscles round the hip-joint have been paralyzed. Should there be much hyperæsthesia here, the resemblance of the case to one of tuberculous hip-joint disease or of osteomyelitis may be close. In this connection it must be remembered that the hyperæsthesia may continue for as long as six weeks after the onset of the disease.

The very severe cases are often mistaken for examples of peripheral neuritis, most commonly of course, of post-diphtheritic paralysis. The onset of the paralysis is, however, different in the two conditions: in acute poliomyelitis it develops suddenly, in post-diphtheritic neuritis it appears gradually, being preceded by a few days' soreness. In the latter, the cranial nerves are very prone to be involved, particularly those of the pupils, eyes and palate, and the heart may show evidence of myocarditis. In acute poliomyelitis the cranial nerve nuclei tend to be spared, and the repurgitation of fluid through the nose is not seen, while the extreme flaccidity of the paralyzed limbs is very characteristic of the disease. In peripheral neuritis the sphincters are not affected, in the severe forms of acute poliomyelitis they may be paralyzed. Landry's paralysis may be suspected, but this disease is practically unknown to occur during childhood.

Course and Prognosis.—As has been already stated, the paralysis reaches its maximum at once, or within a few hours. Except in the

in relapsing cases, the paralysis does not spread, and any change that occurs is towards recovery.

Some of the affected muscles may recover very rapidly, others in the course of weeks or months, while others remain permanently paralyzed; the outlook depends upon the amount of damage to the anterior cornu cells. Certain of them at the onset of the disease lose their function from shock, although but little altered structurally; in such the recovery will be rapid. Other cells are more or less severely damaged, but are able to recover in the course of time; while some are damaged beyond recovery. Complete recovery of all the affected muscles is rare except in very slight cases.

Recovery due to restoration of the continuity of the nervous supply of the muscle must take place within a few months, but it is certain that the functioning of the limbs may continue to improve for two years or even more. This late improvement is probably due, partly to the restoration of tone in muscles which were overstretched when paralyzed, and partly to the limb becoming accustomed to its altered powers. It is also possible that portions of the affected muscles recover and becoming hypertrophied, give rise to improvement in the functioning power of the limb.

Bulldozing of the abdomen due to persistent paralysis of the abdominal muscles is an uncommon fault.

The electrical reactions of the muscles give a fairly satisfactory basis for prognosis, but the outlook is often rather more favourable for the reasons just given, than might be supposed on these grounds alone. If at the end of fourteen days the reaction to faradism is lost, the muscle tested will be paralyzed for a long time, and may be permanently affected, wholly or in part. Where the faradic response remains, although lost later, recovery will occur. Complete loss to faradism and galvanism makes it unlikely that recovery will take place. Where the faradic response remains throughout, recovery will be rapid. A general anæsthetic is usually resorted to for a satisfactory examination of the electrical reactions of the muscles of a child.

Besides the outlook as regards the paralysis, we have to consider the possibility of interference with the growth of the long bones of the limbs. Here we are on difficult ground, and it is to be remembered that in some cases where the paralysis is not severe, the arrest in the growth of the bones may be very marked. Even in slight cases, therefore, this danger cannot be definitely excluded. In severe cases, stunting in the development of the bones is very likely to ensue.

The danger to life is slight. The mortality during the acute stage of the disease has been so small that it has taken many years to determine the exact etiology of the condition. Death may occur from involvement of the respiratory muscles, and possibly from damage to the vital centres of the medulla (p. 215). After the acute stage, where the muscles of respiration have not completely recovered, death is prone to occur from bronchitis or pneumonia.

Treatment.—No prophylactic measures are at present known. During the initial stage of the infection, should the child be fretful or in pain, sedatives should be given. If possible, crying should be prevented, in order to minimize the risk of additional vascular changes in the spinal cord. The paralysis develops so rapidly that any counter-irritation to the spine can hardly be of much value, and may cause the child additional distress. Possibly the administration of iodide may hasten the absorption of the inflammatory products in the spinal cord.

After the initial stage, the indications for treatment are to maintain the nutrition of the affected muscles, to prevent the stretching of the paralyzed muscles, and to encourage voluntary movement of the limbs.

Under the first heading comes the use of warmth, massage—ordinary or electrical—and baths. The paralyzed limb must be warmly clad, and requires more covering than a normal limb. As soon as the hyperæsthesia has passed off, massage should be started. Ordinary massage is usually the best, for it is not generally possible to make a child take kindly to treatment by electricity. Any forms of massage are of use so long as they promote a good circulation of blood through the limbs. The massage should be given twice daily, and must be persevered in for months to even years. It can quite well be done by the mother or nurse. Hot and cold douches to the limb are also of use in increasing the blood-supply of the limb.

Stretching of the paralyzed muscles must be prevented by the use of light splints, which can be easily removed for the massage and exercises. It is very important that no over-stretching of the muscles should be allowed to occur during the first few months particularly, lest the muscles, if they recover, are hindered by having to act at a mechanical disadvantage. Where the anterior tibial muscles are paralyzed, the weight of the bedclothes must be taken off the feet, and the ankle kept at right angles to the leg by means of a light splint.

To encourage voluntary movement is an important part of the treatment so that the child may learn to make the best use of his affected limb. Electricity is perhaps of greater use in this way than in the form of massage, and to flaccidize the child with an electrode attached to a burning battery provides a strong stimulus towards voluntary action. Another plan is to place the child in a bath in order to diminish the weight of the limbs, so that he begins to find that he is able to make some movement of his limbs, and thereafter endeavours to use them more. Many other methods of exercising the paralyzed limbs may be devised in order to encourage the patient to use them, and to strengthen those muscles or parts of muscles which can be used.

The massage and exercises should be continued for many months or even years, in order to get the best possible result.

Later, various surgical procedures may be of use. Tenotomy,

fixation of joints, and nerve or tendon suture, may be of great value. Where mechanical means are taken in order to increase the use of the limb, care should be taken that they allow as full movement as possible to such muscles as are not paralyzed.

XII.—THE INFECTIOUS FEVERS OF CHILDHOOD.

With the exception of diphtheria, these diseases are due to organisms as yet not isolated with certainty. Other points in the etiology are, however, well known, and in the descriptions that follow, under this heading, are given the age and seasonal incidences, the periods of incubation and of infectivity, and the length of time for which a child who has been exposed to infection should be kept in quarantine. The period of incubation is to be regarded as the interval of time elapsing between the infection and the appearance of the first symptom of illness: and not as that between the infection and the development of the rash. With the onset of the first symptom of the disease, the period of incubation ends, and the prodromal stage, or stage of invasion, starts. The incubation period of any of the diseases is subject to variation in different cases.

The universally notifiable diseases in this group are diphtheria and scarlatina. Small-pox, which is of course notifiable, as is typhoid fever, will not be described, except in so far as the differential diagnosis of chicken-pox requires the mention of the chief features of variola. Varicella is in some districts made notifiable during an epidemic of small-pox: and at the time of writing, all cases of meningococcal meningitis in London are required to be notified.

Some of the dates which should be borne in mind in this group of diseases are given in tabular form on the opposite page (Table 20).

Disinfection.—A few remarks under this heading may be of use. The disinfection of clothes and bed-linen may be undertaken by the local sanitary authorities, and is also done by many private firms whose work is very satisfactory. The best method adopted is that involving the use of superheated steam at high pressure, the materials being afterwards dried by hot air.

As regards the disinfection of a room lately occupied by an infectious patient, it must be remembered that dust is the chief source of danger. The best method of disinfection consists in the thorough scrubbing of the entire room and its contents with soap and water. This, however, is but rarely practicable in private houses: but so far as it can be allowed, this washing should never be neglected, whatever other means are employed. Of the gaseous disinfectants, sulphurous acid and formalin vapour are the two most commonly employed. The

Disease		Season	Average Period of Incubation	Average Duration	Time of Eruption	Period of Convalescence
Dysentery	—	Autumn	About 4 days	12 days	—	Until free of bacilli
		Autumn	About 4 days	10 days	2nd day	Until 4 weeks after appearance of rash, if no other changes or alterations
Measles	—	June and Incubative	About 10 days	15 days	2nd or 4th day	Until 4 weeks after appearance of rash, if all other than symptoms have passed
		Spring	About 14 days	3 weeks	1st or 2nd day	Until rash has disappeared, or absence of infectious symptoms
Varicella	—	Autumn	About 14 days	3 weeks	4th or 5th day	Until scabs have fallen off
		Winter	About 14 days	4 weeks	—	Until (all other) symptoms have disappeared (6 weeks)
Measles	—	Winter and Spring	About 11 days	14 days	—	Until end week after feeding has disappeared

Time of Onset refers to incubation and not to infectious period.

former, generated by burning sulphur, cannot be said to be effective, as from lack of oxygen the sulphur ceases to burn before the percentage of sulphur dioxide in the air of the room is sufficiently high to exercise a strong germicidal action. Various forms of apparatus, of which probably Lunge's is the best, are in use for the production of formalin vapour. This method is fairly efficient, but lacks any great penetrating power, so that on dust and upholstered furniture its action is not reliable. The smell of formalin may be removed by placing bowls of liquid ammonia in the room, which forms an inodorous compound with the formalin. Another method, which is well spoken of, and which can be easily carried out, has been invented by Evans and Russell. This consists of adding formalin to potassium permanganate. An energetic reaction takes place at once, and formalin vapour is given off in large quantities. For a small room, an oz. of potassium permanganate should be used, and 1 pint of formalin (40 per cent) should be added to it. An ordinary metal bucket may be used to hold the reagents.

SCARLATINA.

Etiology. The seasonal incidence of scarlet fever shows that it is most prevalent during the autumn and early winter months. The age at which children are most likely to be attacked is from five to ten years, the next most common time being the first quinquennium of life. It is rare in babies. The incubation period is short; usually three or four days (one to seven). A quarantine of a week is probably sufficient in the case of a child who has been exposed to infection; but the Medical Officers of Schools' Association recommends ten days.

In its intensity the disease shows some variation. During the first day it is but slightly infectious; and in a children's ward, if properly ventilated and aired, no harm should arise from the admission of a case in this stage. Later, the infectivity increases, and is particularly connected with the facial mucus, or the discharges from the ears or nose. The desquamating cuticle is probably only dangerous from such contamination, just as the patient's clothes or toys may be. In these ways, as well as by direct contact, the disease may be spread. Outbreaks have been traced to infected milk. The exact period for which isolation should be enforced is not yet quite definitely settled; but it is certain that the faecal, nasal, and ear discharges are a source of danger, and no patient may be allowed to mix with others while these are present. The Medical Officers of Schools' Association advises that isolation should be maintained for "not less than six weeks from the date of the appearance of the rash, provided convalescence is completed and desquamation has ceased, and there is no sore throat discharge from ears, suppurating glands, or eczematous patches." This rule, possibly, errs on the side of over-zealousness; and in most fever hospitals the patients are now sent home after four weeks in the absence of any discharges and of albuminuria.

A second attack of scarlet fever is occasionally met with, but, as a rule, one attack confers immunity for life. A relapse is a very rare occurrence.

Symptomatology.—The pre-eruptive stage does not last for more than twenty-four hours, and the symptoms of invasion are rapid in onset, and often severe in type. The most constant are headache, vomiting, and sore throat. The headache may be associated with pains in the back and limbs. Vomiting is an important sign, and may be continuous, or occur merely once. It is a very constant sign. With it there may be diarrhoea. The soreness of the throat causes discomfort on swallowing; and when inspected, the tonsils, together with the whole of the buccal mucosa, are seen to be swollen and reddened. The glands beneath the jaw are slightly enlarged and tender. The tongue is coated, the temperature high (102° or more), and the pulse rapid.

With the appearance of the rash these symptoms develop further, and are usually at their worst on the third or fourth night of the disease. The child becomes prostrate, perhaps convulsed, often delirious at night. The temperature ranges between 102° and 104° ; and the pulse is often as frequent as 150 per minute. The swelling and injection of the throat increase, the tonsils become covered with a purulent secretion, and after the third or fourth day may show some ulceration. The tongue is at first covered with a white fur, through which the injected swollen papillae appear. Cleaning begins on the second day, the edges and tip clearing first, and being bright red in colour. By the third or fourth day the tongue is all clean, and is red and raw-looking, being denuded of its epithelium. This constitutes the "strawberry-tongue" of scarlet fever. The combination of a clean tongue and dirty throat is very characteristic at this stage.

The rash is bright red in colour, and consists of two elements, an erythematous flush and numerous cloudy-see red papules, giving a punctate appearance to the eruption. It appears first on the neck, chest, and upper arms, and spreading rapidly, becomes fully developed on the third or fourth day of the disease. On the face, the punctate appearance is wanting, and the rash is represented by the flush only, while the circum-oral region is unaffected, and remains pale. The palms and soles similarly show no punctation. The rash is usually best seen in the axillary areas, the nuchal region, and the flexures of the joints.

In the severer forms of the disease, the papules may show minute vesicles on their summits, or they may be petechial. Such modifications of the typical rash are usually seen in the flexures of the joints.

With the full development of the rash, the temperature begins to fall, occasionally by crisis, but more often gradually, and the normal is reached by the end of a week in cases of ordinary severity. The pulse-rate is usually diminished before the fall of the temperature. There is a gradual diminution of all the symptoms. The rash fades

in the order in which it came, and is usually all gone by the end of a week. No molting of the skin is left, but a uniform yellowish staining is seen. The papules remain hot upon the outer side of the upper arms and legs, where the skin is normally harsh, and are often well seen here in the second week of the disease.

The disappearance of the rash is usually followed immediately by desquamation; in fact, on the face a fine powdery desquamation is usually seen before the rash has gone, giving the characteristic "powder and rouge" complexion by the third or fourth day. In infants, the peeling is generally slight and transient. Children with gray skins tend to peel less than those with dry skins. As a rule, desquamation, both in amount and persistence, is proportionate to the severity of the attack. Usually beginning on the third day, it may be delayed for three or even six weeks. On the face, behind the ears and on the inner parts of the arms, the desquamation is powdery; while on the neck, trunk, and limbs is seen the typical "pin-hole peeling," in which the flakes are circular, with a minute central perforation due to the rupture of the epidermis of one of the papules of the rash. Later these scales coalesce, and larger flakes of irregular shape are found. On the hands and feet, where the cuticle is thick, the epidermis becomes dry, shiny, and wrinkled, and is usually thrown off in large shreds, beginning at the tips of the digits.

Desquamation is commonly completed by the sixth week; but, as has been mentioned, may be much retarded.

Peeling occurring on the chest, hands, and feet is very typical of a recent attack of scarlet fever.

Variations in the form of the infection are very common; perhaps no disease is more liable to deviations from its ordinary course.

The simple form of scarletina may be much milder than the type described above; the rash may be slightly masked, and escape observation, and the temperature may not be raised above 100°.

In its more severe forms scarlet fever is becoming less common; the type most frequently seen shows a high range of fever, with delirium, unconsciousness, and persistent vomiting. Death occurs from exhaustion or convulsions within a week. Where much alteration of the throat is present (anginal form), a septic infection may occur. The temperature remains up, and becomes remittent in type; the tongue becomes coated again, and a septic rash appears. There are severe prostration and emaciation, with much pain and ulceration of the throat, accompanied by a lumpy swelling of the tissues of the neck. The constant distress causing loss of sleep and inability to swallow, combined with the severity of the disease, may cause the death of the patient within ten or fourteen days. Other cases show more septicæmic symptoms, sweating, and diarrhoea; and in these death usually occurs within three weeks from septic broncho-pneumonia. Others, again, develop pyæmia, with abscesses appearing in the glands,

joint, pericardium, or pleura. The septic rash is usually morbilliform, and is limited to the cheeks, buttocks, and extensor surfaces of the limbs. It is sometimes only an erythema. The great majority of cases showing a septic rash end fatally.

The form in which the infection is so severe as to cause death before the rash appears ("atrophic form") is very rare.

Complications and Sequelæ.—Exaggeration of some of the morbid conditions in scarlatina accounts for some of the complications. Extensive ulceration of the fauces may give rise to hæmorrhage, or to perforation of the soft palate. The inflammation may extend to the larynx. Rhinitis may occur. Diarrhoea may prove dangerous. Convulsions may be seen in the severe forms of scarlatina, or may arise in connection with nephritis or, rarely, with meningitis.

Other exanthems may co-exist with scarlatina: measles, varicella, small-pox, and typhoid, have all been reported. Diphtheria not uncommonly complicates scarlet fever, and then is usually fatal. Measles and varicella, however, do not make the prognosis any less favourable.

Otitis media arises in nearly 15 per cent of cases (Kager), and may result in intracranial disease or deafness.

Albuminuria and hæmaturia are found in about 8 per cent and 4 per cent of cases respectively. Although nephritis may arise early in a severe attack, it more frequently occurs during the third week, and may follow mild or serious cases. It is said to be most common on the nineteenth day of the disease. It is usually associated with diæsis, and runs a favourable course. In addition, mention may be made of cases of general oedema, in which there is no albuminuria. This condition, which has been termed "simple anasarca," clears up satisfactorily. Its origin is very obscure. In it, the urine is usually decreased in amount.

Arthritis occurs very frequently at the end of the first week as the temperature falls. It is most common in the joints of the fingers, and at the wrist and ankles. In the septic forms of scarlet fever, suppuration may occur. In children's hospitals, ordinary rheumatism is frequently seen following scarlet fever, and shows the typical manifestations, arthritis, heart-disease, nodules, and slight chorea (p. 165). Well-marked chorea is not uncommonly met with as a sequel to scarlatina.

Cervical adenitis from a septic throat infection, usually arises during the third or fourth week of the disease. It is associated with a renewed rise of temperature and, as a rule, with albuminuria. Suppuration may occur. The enlargement of the glands may persist for many weeks.

Mention has already been made of the pyæmic abscesses, the purulent pericarditis, pleurisy, and meningitis, that may arise in connection with the septic form of scarlatina.

A very rare sequel of scarlatina consists in a peculiar and persistent thickening of the skin and subcutaneous tissues, which may develop in the cheeks, buttocks, thighs and elsewhere.

Diagnosis.—The points in the differential diagnosis of scarlatina from rubella and measles are considered in the following table:—

		SCARLATINA	MEASLES	RUBELLA
	Period	1 day	1 day	1 day, or longer
FORWARDING STAGE	Symptoms	Severe: vomiting, high fever, headache, scarlet throat, quick pulse.	Catarrhal: fever, sore-throating, conjunctivitis, laryngitis, diarrhoea. Noddy's spots.	If present: slight catarrhal symptoms.
	Rash, etc.	Appears on neck and chest. Scarlet points in dusky red skin. Clean new tongue by 4th day.	Appears at mouth—face. Dark-red papules only becoming confluent on pale skin. Throat patchy and red.	Appears on face. Pink, small papules; early desquamation. Throat, general redness.
REVERSE STAGE	Urogenital region	Pale.	Discrete.	Discrete.
	Desquamation	Pale, patchy.	Marked, scaly.	Erased: may be typical.

FORWARDING STAGE, THE UROGENITAL REGION; REVERSE STAGE, DESQUAMATION.
SCARLATINA, MEASLES, AND RUBELLA.

Blistered rashes, usually evanescent, various septic rashes, or an eruptive rash, may simulate the rash of scarlatina; but the diagnosis can here be made by the absence of the symptoms of scarlet fever, and the condition of the fauces and tongue. A belladonna rash is very like that of scarlatina; but the history, together with the dilated pupils and possibly very active delirium, serve to differentiate the condition. A rash in acute rheumatism may simulate that of scarlatina, as may that of influenza and the erythema of diphtheria.

In acute tonsillitis there is little or no enlargement of the glands at first; vomiting, too, is absent.

The combination of a dirty throat and a clean red tongue is very characteristic of scarlatina at the third or fourth day.

The condition of the throat may give rise to a suspicion of diphtheria, which is best settled by a bacteriological examination. As has been stated, the two conditions may coexist.

During the first few hours of illness it may be difficult to say if the condition is due to scarlet fever or primary pneumonia. In both

there is a rapid rise of temperature, often with vomiting. The rate and character of the respirations and the condition of the throat, will usually settle the matter in the absence of any pulmonary signs (p. 94).

The circum-oral pallor is by itself of no value in the diagnosis of scarlatina. It is present in many febrile conditions associated with a furred tongue; it may thus be seen at its best in such a condition as appendicitis.

If first seen during the peeling stage, it must be remembered that desquamation is not peculiar to scarlatina, but occurs after measles, rubella and other diseases, and may follow the application of liniments or ointments. Pin-hole desquamation, although very suggestive, is not absolutely pathognomonic of scarlatina, as it may occur after rubella. Most characteristic of the disease at this stage is the peeling at the hands and feet. This may be copious, large portions of epidermis being shed and leaving a thin, shiny pink skin in the denuded areas, most marked at the tips of the fingers. This has to be distinguished from the slight peeling which may occur on fingers that are sucked or feet that are not regularly washed. It may be associated with peeling on the chest, on outer sides of the arms or thighs, or with otitis media, nephritis and other sequelæ of scarlatina. Congenital sublyones is usually to be easily distinguished by the history, the universal distribution of the scaling, and the thickening of the skin.

Prognosis.—The mortality statistics of scarlatina show that the younger the child the more dangerous is the disease to life. Sufficient allusion has already been made to the symptoms which point to a severe infection. The influence of the co-existence of other co-infections with scarlet fever has also been mentioned.

Treatment.—In mild cases, little treatment is demanded beyond isolation in a well-ventilated and warmed room, with tepid sponging night and morning. The diet should consist chiefly of milk, to which jellies, broth, and oranges or grapes may be added. With a mild infection, the child may be allowed to get up after ten days or a fortnight, and to go out of doors, if the weather be warm, a few days later. Care must be taken to prevent chills.

When the throat symptoms are mild, the child may be given ice to suck, and should have the throat sprayed with some antiseptic lotion. Painting the throat with a glycerine preparation, such as that of boracic acid, is usually comforting. Where the glands are tender, they should be dressed with fomentations.

If the faucial condition is severe, nothing is preferable to douching with chlorine water. The solution is made by adding strong hydrochloric acid (HCl) to powdered potassium chlorate (p. 181), and to this water up to six ounces is gradually added, with frequent shaking. At the time of use an equal quantity of hot water is added. About half

a pint should be used on each session. If possible, the child sits up with his head bent forward over a basin. The lotion is gently introduced into the mouth from an enema rubber syringe, time being allowed for the patient to breathe between each squeeze of the bulb. If the child is lying down, the tube may be introduced between the teeth and the cheek. In this way the fauces should be washed every three to four hours. Similarly, by introducing the tube into the nostril, the nasal passages may be cleansed if necessary, the child being instructed to keep his mouth open, in order that the fluid may return through the opposite nostril. The same lotion may be used, but the syringing should be more gently performed. In young children the discomfort and difficulty connected with swallowing may necessitate tube feeding. When desquamation is proceeding, a hot bath should be given daily. Vaseline or carbolyzed oil may be applied.

The complications need not here be treated in detail. A non-purulent arthritis should be treated by salicylate. With the obvious exception of the avoidance of chills, we know no means—either by diet or enforced rest in bed—which will prevent the onset of renal mischief.

Bearing in mind the great liability to post-scarlatinal rheumatism, the condition of the throat after an attack of scarlet fever should be watched. Should there remain any tendency to a chronic tonsillitis, this must be treated and, if necessary, the tonsils should be removed.

MEASLES (*Morbilli*).

Etiology.—The *seasonal prevalence*, or more accurately, the mortality incidence, shows two maxima in the year—June and December. The disease is common amongst children of any age. The incubation period is almost invariably ten days (seven to fourteen). Quarantines should be for sixteen days. The patient is infectious from the very beginning of the illness until at least a fortnight after the appearance of the rash, and then only may be regarded as safe if all catarrhal symptoms and desquamation have ceased. A second attack of measles is not very uncommon.

Conditions associated with catarrhal states of the respiratory passages, such as whooping-cough, and tuberculosis, predispose towards measles, as do the other infectious diseases.

Symptomatology.—The symptoms of invasion usually develop rapidly. The temperature runs up to 102–3°, and is associated with loss of appetite, headache, and a tired feeling. Vomiting is rare, though nausea is not uncommon. The catarrhal signs are marked. Infection of the conjunctivæ, watering of the eyes and nose, sneezing and coughing from tracheitis and bronchitis are very constant. Hoarseness may be present. Diarrhoea is common at this stage. Photophobia may give rise to much discomfort. A blotchy, erythematous rash occurs sometimes on the face and chest. Koplik's spots are seen in the

pro-eruptive stage in most cases, fading rapidly with the appearance of the rash. They are minute, bluish-grey spots on a reddened base, appearing first on the buccal mucosa opposite the lower molar teeth, but later in any position on the lining membrane of the cheeks. They are better seen in diffused daylight than in artificial light.

Mention should, perhaps, be made of cases—relatively few in number—in which some malaise and loss of appetite are present throughout the incubation period. Such a condition, seen occasionally in most of the acute specific fevers, is perhaps least uncommon in measles.

After forty-eight hours in mild infections, the symptoms begin to cause less discomfort, and the temperature becomes lower. In severe cases no such improvement occurs, but in either case, with the appearance of the rash the fever is at its highest, and the catarrhal symptoms at their worst. The eruption is first seen on the third, or more usually the fourth day, and with its advent rapid improvement is the rule. The temperature rarely remains high for more than thirty-six hours, and when the rash is fully developed the temperature falls quickly. The normal is reached on about the sixth day if no complications are present.

The rash appears first on the forehead at the margin of the scalp and behind the ears. It spreads quickly on the face, involving usually the circum-oral region, although here there may be no more than a blotchy condition of the skin. From the face it spreads rapidly to the trunk, where it is best seen, and to the arms and lower extremities. Both the flexor and extensor aspects of the limbs are affected. The palms and soles do not, as a rule, show more than a flushed appearance, but here, even if spotty in character, the rash is never missed. The palms, or palmar area, may be affected very early.

The eruption appears first as small red spots, which become rapidly larger and distinctly papular. The papules tend to coalesce quickly, forming irregularly-shaped injected areas raised on a background of pale unaffected skin. The rash can often be seen early on both hard and soft palate, as dark red patches.

The rash rarely remains in any part for more than twenty-four hours, and fades in the order in which it appeared, so that by the time it is fully developed on the legs the face has cleared. By the end of the week the rash will have disappeared, leaving a brown mottled stain. The fading of the rash is usually followed by a fine branny desquamation, which may last for ten days. The peeling is rarely extensive, and may be found confined to the trunk, or to the face and limbs. It is not seen on the palms or soles.

Variations of the form of disease are sometimes seen. The infection may be so mild as to be unaccompanied by an eruption. Of severe cases, two varieties are recognized. The pulmonary form shows early and severe pulmonary disease, with dyspnoea, cyanosis, and high fever. The rash may be suppressed. In the toxic form there develops a typhoid state, delirium, tremor, feeble pulse, and high temperature.

The rash is intense, and becomes petechial. Epistaxis is common. In both these forms death usually occurs within a week.

Measles following scarlatina tends to be severe in type, and often the pre-eruptive stage is shortened to one day or less.

Complications.—Exaggeration and extension of the initial catarrhal symptoms account for the majority of the complications. Of these, acute laryngitis, otitis media, severe bronchitis, and bronchopneumonia are the most common. Catarrhal stomatitis may be found, which in emaciated children may show gangrenous changes. Carcinoma and *rosita pudendi* are rarely seen apart from measles. Catarrhal enteritis may cause fatal diarrhoea. The affection of the eyes may predispose to pterygoid conjunctivitis; or a purulent condition may develop, with ulceration of the cornea, hypopyon, or panophthalmitis. A macropapular dermatoma may arise from the initial nasal catarrh, and by extension along the Eustachian tubes may cause otitis media.

Sequelæ.—A notable irregularity of the heart's action occasionally develops after measles, and may persist for two or three months; but it does not, as a rule, produce any dangerous symptoms. True rheumatism is a rare sequel.

As in the case of whooping cough, measles may be followed by tuberculosis disease in the lungs or subintestinal glands. In any case in which the symptoms would seem to point to such a condition, care must be taken to exclude the presence of a latent ringworm. Where active tuberculosis is present, measles exercises a baneful influence on the condition.

Diagnosis.—The points in the differential diagnosis of measles from rubella and scarlatina are tabulated on page 225.

Prognosis.—Measles is most fatal between the ages of six months and two years. From this age onward the mortality rapidly lessens, until, after the fifth year of life, measles very rarely causes death. Unfavourable symptoms are the severe pulmonary or toxic symptoms already enumerated, or those due to complications.

Treatment.—Little need be said under this heading, for the treatment consists chiefly in isolation, and in endeavouring to ward off any complications which may develop. The photophobia may be distressing, and necessitate shutting the light in the room. The bronchial or intestinal catarrhs must be treated in the ordinary way. Laryngitis may be relieved by the use of a steam-bottle. Intubation is occasionally necessary. The mouth and nose must be carefully cleaned in order to prevent oral complications.

Owing to the danger of inducing diarrhoea, the customary initial purge is better omitted.

In older children, who are usually very much depressed by the discomfort of the disease, books and toys should be allowed. Toxic symptoms may call for the use of stimulants and sedatives. Every effort should be made to secure a complete and rapid recovery. The appetite should be stimulated, and iron with cod liver oil given for the anemia, if such measures are necessary. Should any enlargement of glands occur, or complete recovery appear to be retarded, a visit to the sea or country should be suggested.

RUBELLA (*German Measles*)

Under the names of German measles, röteln, rubiola, and others, rubella has gradually become universally recognized as a clinical entity separate from scarlatina and measles. It was first accurately described in England by Maiten in 1814. While at one time there were many who denied the existence of such a disease, there are now some who hold that under this name are described two conditions, one of which is truly rubella, the other not. This view, which is not generally accepted, is most prominently upheld in this country by Dr. Dukes, who claims to have separated from rubella an infectious disease which he calls "the fourth disease," since it is neither rubella, scarlatina, nor measles.

Etiology.—The *age-incidence* of the disease shows one peculiarity in that, although it is most common in young children, yet it occurs in older children and adolescents far more frequently than does measles, presumably because the majority contract measles in early life, owing to its great infectiveness. Rubella has been reported in an infant a few days after birth (Scholl).

Its *seasonal incidence* shows that it is most common in the spring months, three-quarters of the cases occurring between March and June, with a maximum in May. The incubation period is long, from ten to twenty-one days, usually from fourteen to eighteen. Quarantine, where necessary, should be for three weeks.

Symptomatology.—Symptoms of invasion are absent in the majority of cases and the rash is the first thing noted, as in varicella. In some cases there are sore throat, moderate fever, slight coryza, and enlargement of the glands in the neck, with occasionally pain in the neck. Vomiting, it should be noted, is "exceedingly rare" (Kluger). These symptoms do not precede the appearance of the rash by more than twenty-four hours. The throat shows a general redness. During the first twenty-four hours of illness small bright red points may be present on the uvula and soft palate (Förchheimer).

The rash at first rather resembles that of measles, changing by the second day to one which is more scarlatiniform in character. It rapidly disappears in three or four days. It is very characteristic of the eruption that it appears on the face and rapidly sweeps down the length of the body. In any one place it is not visible for more than twenty-four hours.

It appears usually first on the face, in the form of pale pink discrete spots which are paler, pinker, and more discrete (early) than those of measles. There is no circum-oral region left free of the eruption. On the trunk the rash usually becomes confluent, so that a uniform erythema, sometimes puritate, is present. As the face is now clear again, the case resembles strongly one of mild scarletina. The rash now usually disappears rapidly, and does not last for more than three days. In exceptional cases the rash on the body may remain discrete or may become confluent, forming macules which are smaller than those of measles. Rarely, the rash does not appear on the face at all, but is first seen on the trunk and limbs as a scarlatiniform erythema. This form, with some mild cases of true scarlet fever, is generally held to constitute what Dr. Dukes describes as "the fourth disease." The fever is very mild in amount and short in duration—hardly over 100° or 101°; it lasts not longer than twenty-four hours, and is present usually while the rash is coming out.

Glandular enlargement is present in the majority of cases; usually the posterior cervical and mesial glands are affected, but sometimes other superficial glands may become swollen. Rarely, glands other than the cervical are the only ones affected. In some epidemics the frequency of the adenitis is less than in others. The glands, when swollen, remain discrete and never suppurate.

Catarrhal symptoms are very slight. Frequently there are some itching of the eyes, lachrymation, and a little secretion of the conjunctivæ. Sneezing and a slight watery discharge from the nose may also occur.

The course of the disease, as has been indicated, is very rapid, the rash rarely lasting for more than three days. In any one place the rash lasts for twenty-four hours only.

Desquamation. Although it may be absent, is commonly unprofuse than in measles, and may be very copious. It takes a fine-beamy form, but it appears undoubted that "pin-hole" desquamation may occur.

Infectivity.—*Rothelia* is certainly contagious for a day or two before the onset of the rash and while the rash is appearing, but at no time is it very contagious. By the time the rash has disappeared, unless catarrhal symptoms persist, the patient is no longer infectious at all. It would seem that if personal disinfection be carried out under ordinary circumstances the patient may be regarded as safe after a week.

Complications.—Occasionally two or three days after the rash has disappeared, there is a superinfection of it in its morbilliform, or scarlatiniform character. Otitis media has been reported.

Diagnosis.—The chief points concerned in the diagnosis of rubella from scarlatina and measles are given on page 228.

Prognosis.—This is almost invariably good. Some epidemics are more severe than are others, but death hardly ever occurs in this disease.

Treatment.—Isolation and rest in bed comprise the only necessities of treatment in most cases. The patient may be allowed out of doors while desquamation is proceeding.

DIPHTHERIA.

This term is confined now to those conditions due to the Klebs-Löffler bacillus, first described by Klebs in 1883.

Etiology.—Diphtheria differs from the other diseases classed as the acute specific fevers of childhood, in that its bacteriological cause is known. Like typhoid, scarlet fever, and rheumatism, it is more prevalent during the autumn and early winter months. The *epidemiology* shows that it is more common in children under five years old than during the next quinquennium. Three-fourths of all cases of diphtheria are found in children under the age of ten years. The period of incubation varies from one to eight days, but is most commonly not longer than from three to five. Quarantine should be maintained for twelve days.

Infection may be transmitted directly, or indirectly by the use of infected material or utensils. Milk is a common channel of infection. Overcrowding at home and at school are potent causes for the spread of the disease. Defective sanitation is probably not directly responsible for the production of diphtheria, but by its effect in lowering the resistance of the body, may tend towards the spreading of the infection. It must be remembered that cats and probably birds may suffer from and transmit the disease.

The *period of infectivity* is to be judged by bacteriological examination; and so long as any diphtheria bacilli are present, the patient must be regarded as a possible source of danger to others.

Clinical Bacteriology.—So many institutions are now prepared to undertake the examination of throat swabs for the Klebs-Löffler bacillus, that there is seldom any difficulty in getting an expert opinion; but in order that the most accurate results may be obtained, the practitioner taking a specimen from the patient's throat must

attend to the following points. In the first place, the swabbing should be taken from the surface of the membrane, if possible. secondly, several hours (here at the least) must be allowed to elapse between the last application of any germicidal lotion to the throat and the taking of the specimen. and thirdly, the apparatus used for the swab, both wool and holder, should have been previously sterilized, and be so protected as to reach the bacteriologist's hands without further contamination. Should these points be overlooked, various difficulties will arise. firstly, diphtheria bacilli, although present in the throat, may be missed on bacteriological examination. secondly, they may be recognizable in a smear preparation, but, being dead, they cannot be cultivated. or, thirdly, there may be an exuberant overgrowth of contaminating organisms, so that the Klebs-Löffler bacillus cannot be distinguished on culture. It is evident, therefore, that should the practitioner neglect the points enumerated here, the bacteriologist's opinion may become of no value, and dangerous mistakes may be made.

In taking a specimen from a throat, any form of home-made apparatus is better avoided, and those forms sent out from bacteriological laboratories are the most suitable for use in all cases. They consist of small air-tight cases of metal or glass containing some cotton wool on a holder, all of which have been sterilized by dry heat. In use the holder is withdrawn from the case, the wool rubbed gently over the membranous exudate, and then replaced in the case at once. The apparatus is not opened again until the examination can be made.

It is not possible to give here the details of the bacteriological methods to be employed in the examination of the specimen. These the reader may obtain in works on clinical bacteriology. Should the material be sent, as is usually advisable, to a laboratory, it should be borne in mind that a positive diagnosis cannot be made from an examination of a smear preparation only, and it is necessary for a culture to be examined before certainty is reached. It is sometimes possible for the diphtheria bacillus to be recognized after incubation for so short a time as six hours; but in the majority of cases twelve to eighteen hours' growth is necessary.

It must be remembered that the Klebs-Löffler bacillus may be found in the throats of healthy persons; but such individuals must be regarded as a source of danger to others, particularly to children with any form of tonsillar disease.

Morbid Anatomy.—The membrane consists of a network of fibres enclosing epithelial and small round cells in its meshes. The bacilli are found in the deeper layers, while in the superficial layers of the membrane are large numbers of cocci. The subjacent epithelial cells show degenerative changes, with fragmentation of their nuclei, and there is leucocytic infiltration of the mucous membrane of the affected part. The glands in the neighbourhood of a diphtheritic exudate are invariably enlarged.

The visceral lesions found in diphtheria are due to the toxins manufactured at the site of the membrane. Degeneration, similar to that found in the cells below the membrane, occurs in the liver, spleen, and kidneys. The myocardium shows many changes, the most important of which is the fatty degeneration of the muscle, which may proceed to actual destruction of many of the fibres. Irregular areas of the myocardium, chiefly in the wall of the left ventricle, are thus damaged, the destruction of the muscle fibres being far greater in amount than in the case in rheumatic myocarditis. The fibres of the diaphragm also show a fatty change.

The peripheral nerves show a patchy degeneration, and in severe cases secondary cood lesions are present.

The blood in all bad cases shows anemia, both the number of red cells and the hemoglobin being reduced. A slight polymuclear leucocytosis is present.

Symptomatology.—The symptoms of diphtheria depend partly upon the absorption of the toxins produced by the organism, and partly upon the localization of the membrane.

The constitutional symptoms are very varying in their severity, and in their mode of onset. They are particularly severe where there is a large area affected from which absorption of toxin can occur, as in faucial cases with extension to the nose or larynx.

As a rule, they come on rather gradually: the child complains of having a sore throat, loses its appetite, and becomes listless, languid, and pale, often rather grey in the face. The temperature is raised, but is generally never 103° than 104° . In about half the cases there is a slight albuminuria. Vomiting is a common initial symptom.

While this is, perhaps, the most usual sequence of events, it is not uncommon to see children suffering from diphtheria in whom the initial constitutional symptoms are very slight. The child may be said to be suffering from loss of appetite, cough, disinclination to play, swollen glands or stiff neck, and when seen, it is found to sit up well, to have fair colour in the cheeks, to rise of temperature, and it is only by the routine examination of the throat that the disease is discovered.

On the other hand, in severe types of the disease the child is rapidly overcome by the toxæmia, showing great prostration, an ashy pallor, a feeble, rapid, and irregular pulse, and a subnormal temperature. Great restlessness or delirium is present, and vomiting and diarrhoea add to the collapse.

There is, then, every variety in the severity of the constitutional symptoms that may be found in diphtheritic cases, but it is an important fact, that where present they tend to assume the type associated with collapse rather than the more fond symptoms of pyrexia.

The constitutional symptoms may be expected to become most marked about the fifth day; but in severe cases, by the third day the

toxicemia may produce great pallor; a thin, irregular pulse; much albuminuria, and drowsiness alternating with restlessness and delirium. Diarrhœa and vomiting may also be present.

It must be emphasized, that in children no case of diphtheria should be regarded lightly, for in those that at first appear to be mild, serious symptoms may arise.

FAUCIAL DIPHThERIA.

Typically, the membrane appears upon the tonsils and rapidly covers them. It is grayish-white in colour, and its margins may be clearly defined against the hyperæmic mucous membrane of the fauces. As a rule, it is rather firmly adherent. At first the membrane appears as a thin white film; later it becomes thick and yellowed. It may show an extension on to the soft palate. In a few cases the uvula is the first part attacked. Often, however, the appearance of the fauces is by no means suggestive of diphtheria, and the patchy distribution of the membrane round the edges of the crypts may resemble strongly a follicular tonsillitis. The glands in the neck are almost invariably enlarged and tender, and stiff neck may be a prominent symptom. The smell of the breath in diphtheria is very characteristic. In bad cases there is considerable pain in swallowing, and where the disease spreads to the nasal cavities, and the larynx, the patient's state is one of great distress.

Under the influence of treatment by serum, the membrane should cease to spread in twenty-four hours, and should shortly begin to disappear.

Diagnosis.—The diagnosis of faucial diphtheria may be of great difficulty, and only to be made with certainty by a bacteriological examination, for cases which bear little clinical resemblance to a diphtheric throat may, nevertheless, be due to the diphtheria bacillus. While the bacteriological examination is of the utmost importance and the ultimate criterion, a few clinical points may be of some assistance. The onset of the illness in diphtheria tends to be more gradual and quieter than in follicular tonsillitis; the temperature is lower. Albuminuria is about equally common in both conditions. Where the membrane spreads on to the soft palate or uvula, the condition is almost certainly diphtheria. In follicular tonsillitis the exudate is always confined to the surface of the tonsils.

The exudate in follicular tonsillitis is easily removed by scraping, and is renewed within the course of an hour or so. In diphtheria the membrane is more adherent, and when it is removed a bleeding surface remains which does not usually again become covered by membrane for several hours. In tonsillitis, the glands at the angles of the jaw may or may not be enlarged; in diphtheria such enlargement is practically invariable, and may be extreme.

The early stage of Vincent's angina resembles diphtheria almost exactly.

NASAL DIPHTHERIA.

Cases in which the nasal cavities alone are infected are usually of a mild type. The cause for severity which the nasal cases have acquired is due to the fact that most of them arise in association with the worst facial forms.

In the mild form, the so-called membranous rhinitis, there is an *æd* nasal discharge, which may be blood-stained, and which contains the diphtheria bacilli. It causes soreness round the nose, and some blockage of the nostrils. There is tenderness of the lymphatic glands beneath the jaw. In such cases the constitutional symptoms are usually slight, and there is very little danger of cardiac or nervous sequelæ.

In the severe cases, where the nasal infection is associated with facial and possibly laryngeal diphtheria, there is so large an area from which absorption is taking place, that poisoning and collapse are present to a marked degree. In these, the nasal discharge is thick and blood-stained, and often contains portions of membrane. Nasal respiration is greatly interfered with, and the distress of the patient is very severe.

The diagnosis of nasal diphtheria is to be made by bacteriological examination of the nasal discharge in the absence of facial involvement. A specimen should be obtained by passing a sterile platinum loop into the nasal cavity under the inferior turbinated bone.

LARYNGEAL DIPHTHERIA (*Membranous Croup*).

When the larynx is affected, it is usually secondary to facial diphtheria; but primary laryngeal cases are not very uncommon. At any time during the first week of facial diphtheria, but rarely at a later date, the larynx may be invaded. When this occurs, there is a gradual development of cough and hoarseness. Within twelve to twenty-four hours, inspiratory and often expiratory stridor are heard. Complete aphonia may develop. The deep glands on each side of the larynx can be felt on careful examination to be enlarged. As the laryngeal obstruction increases in severity, the child becomes very restless and in terror of suffocation. The face is moist, and of an ashy grey colour; cyanosis begins to develop. The obstruction to respiration is shown by the withdrawing of the lower lip during inspiration; and later, by the inspiratory recession of the epigastrium and lower ribs. The cyanosis, restlessness, and movements of the thorax are the guides to the amount of obstruction present; but it is to be noted that these are only trustworthy if the child is lying quiet and undisturbed in bed, so the dyspnoea may be very considerably increased in severity under the stress of fright.

In this form of the disease there is great danger of extension of the membrane downwards; and in most fatal cases, the mucosa of the bronchi is affected, and the lungs show areas of hæmorrhage and collapse.

The diagnosis of a secondary laryngeal diphtheria is, as a rule, easy; the involvement of the larynx being shown by the development of stridor. It is not, however, uncommon in hospital practice, to see the patient for the first time when the laryngeal symptoms have become severe and the fauces are almost clean. Here the diagnosis may be of considerable difficulty. It is to be remembered that a child suffering from stridor, and showing any remains of exudate upon the tonsils, is almost certainly the subject of laryngeal diphtheria. Scalding by the drinking of hot water may produce a similar condition, but is not likely to lead to confusion, owing to the history of the case. Enlargement of the cervical glands, or of those about the cricoid, will be greatly in favour of the laryngeal condition being due to a secondary diphtheria, rather than to a catarrhal laryngitis.

Primary laryngeal diphtheria is far from easy to diagnose, and has, as a rule, to be differentiated from catarrhal laryngitis. In diphtheria, the onset of the stridor is rather more quiet and gradual than in the catarrhal condition; and the constitutional symptoms, if present, are those of collapse and pallor. The temperature is not so high in diphtheria as in the simple laryngitis. Of greatest clinical importance is the enlargement of the deep glands by the side of the cricoid cartilage, which is in favour, in acute cases, of the inflammation being diphtheric in origin.

The laryngitis of measles, retropharyngeal abscess, and a foreign body impacted in the larynx or oesophagus, must be borne in mind as the possible causes of the condition.

In taking a specimen for bacteriological examination, the swab must be passed down the back of the throat as far as possible. A negative bacteriological result is not always trustworthy in excluding the possibility of a primary laryngeal diphtheria. If any suspicion as to the nature of the infection remain, the examination should be repeated.

OTHER FORMS OF DIPHThERIA.

The Klebs-Löffler bacillus may produce membrane in various other parts of the body, and absorption of toxin from such lesions may occur. The dactis may be found upon the external genitalia, the conjunctive, the lips, and the mucous membrane of the mouth. The membrane may extend from the fauces down the oesophagus into the stomach. Occasionally, the diphtheria bacillus is found growing upon an open wound.

Complications and Sequelæ of Diphtheria.—**Albuminuria** is present in about half the cases of diphtheria. As a rule it occurs towards the end of the first week or the beginning of the second, and ceases shortly after the disappearance of the membrane from the throat. Although it may persist for several weeks, diminution in the amount of urine passed, or the appearance of dropsy, are very rare. Complete

recovery is the rule. Hematuria is rarely seen, and should it occur, is very unlikely to be followed by any permanent damage to the kidneys.

Post-diphtheritic Paralysis.—This, which is due to a paralyzation of peripheral neurites, may occur from one to six weeks after the onset of the disease, but most frequently arises during the second or third week of the illness, especially after the tenth day. The first symptom usually noticed—if, as is not uncommon, the child is not confined to bed—is a gradual loss of the power of walking; one of the many conditions in which a child is said to have "gone off his legs." Numbness and tingling of the extremities may precede the onset of the weakness, but these sensory disturbances are rarely complained of by children. Soon after these symptoms arise, involvement of the cranial nerves may become manifest; usually from the fact that the voice acquires a nasal tone, the speech becomes indistinct, and there is regurgitation of fluid food through the nose. These symptoms are due to the paralysis of the soft palate. The nerves supplying the muscles of the eye are frequently affected. Paralysis of accommodation is very common, and with retention of the light reflex is extremely suggestive of the condition under consideration. Older children may complain of being unable to read. A squint or partial ptosis may also be seen.

In more severe cases, a dangerous group of symptoms may arise in connection with the affection of the lower cranial nerves. When there is paralysis of the adductor muscles of the larynx, causing complete aphonia, with which is often associated anesthesia unless care is taken the child may be asphyxiated by food passing into the larynx. Should this happen, the cough reflex being abolished, the child may die at once of suffocation, or later of a septic broncho-pneumonia. Matter vomited from the stomach may produce the same fatal result.

Weakness of the muscles of the neck and trunk may be seen; but more commonly the respiratory muscles, either the intercostals or the diaphragm, are affected. Such paralysis is usually preceded by gradually increasing weakness, so that no sudden onset of urgent dyspnoea calls attention to the condition. Paralysis of the diaphragm is the commoner form, and is shown by excessive movement of the chest, the abdominal wall being drawn in during inspiration, and protruded during expiration; that is to say, the normal movement of the abdomen is reversed. With paralysis of the intercostals, the abdominal movement is abnormally great, but similar in rhythm to that seen in health, while the lower ribs are dragged in by the diaphragm with each inspiration. When the respiratory muscles are attacked, the condition is one of grave danger, and death may ensue from extension of the paralysis.

Tibial Crisis.—Under this name Dr. L. Guérin has called attention to sudden attacks of hither paralysis which may occur in cases of post-diphtheritic paralysis. Premonitory symptoms may be present. They consist of restlessness, pallor, rapid pulse and sighing respirations, the tibial involvement showing itself particularly in the feeble, hoarse,

nasal voice, weak cough, and difficulty in swallowing which leads to an accumulation of mucus in the air-passages. Suddenly all these symptoms become much exaggerated, and the patient appears *adverso*, propped, apyrexial, pale and sweating. The attacks, which may last for a few minutes to some hours, may be brought on by exertion or emotion, but often are not traceable to any particular cause. They are probably due to the action of toxins upon the bulbar centres. They are extremely serious, and although a first attack is seldom fatal, some recurrence, towards which there is a great tendency, usually causes death.

As a rule, post-diphtheritic palsy is seen after the severer forms of the disease; but at the same time it must be admitted that it is not uncommon to meet with slight cases in which no history of diphtheria can be obtained. In this connection it is well to bear in mind that the commonest cause of loss of the knee-jerks in a child is the condition under consideration. The loss of the knee-jerks is very constantly present in these cases, but is not necessarily an early sign; at first, indeed, there may be an increase in the patellar reflexes.

The *Diagnosis* in the majority of cases is easily made; but when doubt arises, the loss of power of accommodation, the paralysis of the palate, together with, possibly, the signs of diphtheritic myocarditis, are usually sufficiently distinctive. Very severe forms of paralysis may simulate acute poliomyelitis, but here the onset of the paralysis is sudden, and not preceded by any stage of weakness, while the cranial nerves in that condition are likely to escape (p. 219).

The *Prognosis* as regards life depends upon the condition of the respiratory muscles and the heart. Should the patient live, complete recovery always occurs; but although the function of the muscles is restored in the course of a few weeks, it may be months, or even years, before the deep reflexes return.

Cardiac Complications.—How far there is a toxic factor in the production of the cardiac condition seen in diphtheria is a matter of some doubt; but it seems certain that the fatty degeneration of the heart-wall is the more constant factor at work. The heart is conspicuously enlarged, the dilatation occurring chiefly in the left ventricle; and such excessive dilatation may occur, especially after exercise, that sudden death ensues. On examination of the heart, together with the increase of the deep cardiac dullness, the heart-sounds are feeble, he-he-art type, and often irregular in rhythm. The first sound at the apex is generally reticulated; and the second sound at the base acquires a somewhat booming character, probably due to dilatation of the aorta. In addition, the rate of the heart-beat may be greatly increased. Bradycardia is less common, but a sign of graver import than is tachycardia.

With such conditions as these, pallor, dyspnoea, and vomiting of cardiac origin, may be added to the patient's symptoms.

Broncho-pneumonia, as has been mentioned, may occur from extension downwards in laryngeal cases. Such a complication is as a rule

fatal; and diphtheritic broncho-pneumonia is the most dangerous form of that disease which occurs in children. Extension to the lungs is almost invariably found in fatal cases of laryngeal diphtheria, although it may have been impossible to detect it during life.

Relapses may occur after the third week. They are less severe than the original attack.

Scarlatina, it should be remembered, is especially prone to follow diphtheria.

Prognosis.—The death-rate is subject to considerable variation in different epidemics of diphtheria, and depends to some extent on the number of young children attacked, and their hygienic surroundings.

The most dangerous cases are those in which the membrane extends from the fauces to the nose or to the larynx; for in these there is a large surface from which absorption of toxin may occur. The signs of profound toxæmia, such as ashly pallor, a normal or subnormal temperature, hæmorrhages from the mucous surfaces, or the sudden onset of severe albuminuria, raise the outlook to be extremely bad.

As a rule, the younger the child the greater the danger; but the prognosis depends also upon the general state of nutrition of the patient.

Another very important point is the length of time which has elapsed since the onset of the illness before the patient comes under treatment. In cases where antitoxin is given on the first day of the disease, the death-rate is reduced almost to zero, and it increases rapidly, the longer the treatment is delayed.

Treatment.—As regards prophylactic treatment—the close inspection of children attending school, together with the prompt isolation of cases of diphtheria, are the most important points. Should a child be known to have been exposed to danger of infection, it must be isolated, and antiseptic lotions should be applied to the throat. The prophylactic use of antitoxin is widely recommended, but whatever value it may have in this way must be due to some antibacterial substance other than the antitoxin itself. The dose given for this purpose is usually 300 units.

The most important point in the treatment of diphtheria lies in the administration of antitoxin; and for the best results to be obtained, the serum must be injected in sufficiently large quantities, and at the earliest possible moment. Should diphtheria be suspected, it is best to give a dose of serum while waiting for the result of the bacteriological examination. In every case of diphtheria in a child, serum should be given; for however mild the infection may appear at the onset, it is never safe to assume that it will remain so.

For the dosage, the following rules may be given as rough guides:—
(1) For an infant under two years of age, the minimum dose is 2,000 units; for an older child, the minimum dose is from 4,000 to 8,000

units, according to age: (2) An extra thousand units should be given for each day that has passed since the disease declared itself. (3) Where there is a large area covered by membrane, as in secondary nasal and all laryngeal cases, large doses are especially indicated. (4) If the growth of membrane is not arrested within twenty-four hours of the injection, a similar, or even increased, dose should be given. (5) Half doses may be administered daily, until the membrane has definitely begun to shrink.

The injection is best given under the skin of the back or loin. Special antitoxin syringes are made, but should they be used it is advisable to attach the needle to the syringe by flexible rubber tubing, so that no danger of wounding the child's arm, should struggling occur.

Combined with the use of serum, local measures should be adopted, and the throat or nose should be syringed out every two or four hours, as is described under the treatment of scarlet fever (p. 220). The serum, by virtue of the antitoxin it contains, neutralises the poisonous products of the organism which are poured into the system, so keeping the patient alive while he can manufacture his own bactericidal substances. The local antiseptic measures should be used in order to check the multiplication of the organisms.

The serum may produce, after a week or ten days, pains in the joints, or a skin eruption of a morbilliform or articular character. Some have held that the increased incidence of post-diphtheritic paralysis observed nowadays forms an argument against the use of the serum, except in laryngeal cases; but when it is remembered that paralysis is apt to follow the more severe forms of the disease, it may well be believed that the increase in the number of cases of paralysis is due to the fact that many who formerly would have died, now recover under the use of the serum.

In laryngeal cases, in addition to the injection of full doses of antitoxin, in the early stages a steam-kettle may be provided. Where the dyspnoea is severe, and the amount of obstruction is to be recognised by the signs already given, tracheotomy may be undertaken without undue delay. Intubation in many cases is of great service; but it is not to be recommended unless the patient is in a hospital, so that assistance can be obtained immediately if the tube is expelled. Where tracheotomy has been done, every effort should be made to get rid of the tube as soon as possible, to lessen the liability to subsequent stricture. Much difficulty may be encountered in this, as the removal of the tube causes the child to become very frightened. In order to obviate this a nurse should always be ready to amuse the child with toys and games while he is awake.

Nutrition.—The symptoms of paralysis require special treatment. Where the palate only is affected, thickened fluids may be more easily swallowed than milk alone; or the child may be fed from a feeding-cup, to the spout of which is attached a short indiarubber tube, so that the fluid is delivered into the pharynx. Where there are laryngeal

paralysis and anæsthesia, special precautions must be taken, and it is advisable to adopt nasal or œsophageal feeding entirely. Further, in such a case, should there be any tendency to vomiting, the foot of the bed should be raised on blocks, so that any material vomited may run freely out of the mouth, and not pass back into the larynx.

Strychnine is the most useful drug for the nervous, and should be given in full doses. It may be combined with belladonna or atropine, as recommended by Dr. Lees. Massage may also be ordered for the limbs if they are not tender, and if the condition of the heart does not contraindicate such a measure.

For the relief of strychnine is of particular value, and should be given hypodermically in full doses, together with strychnine.

Cardiac Complications.—Prophylactic treatment, a matter of prime importance, is considered in the next paragraph. Should symptoms arise absolute rest is the first essential, and the child should be kept lying perfectly flat in bed. The various cardiac stimulants, particularly strychnine and atropine, should be given. Some have objected to their use on the grounds that to increase the power of the heart's contraction is but to wear out what little normal muscular tissue remains. To this it may be answered that frequently the condition is one of extreme gravity, and that even a temporary beneficial action may be of the greatest service. It must be remembered that when stimulants are being used over a long period, we are increasing the blood-supply to the cardiac muscle as well as the power of the heart-beat, and that in this way much more than temporary benefit may be obtained. Brandy also appears to do good, although its use is open to the objection that it may increase the possible septic factor in the production of the cardiac condition. In any case it is not of such service as are strychnine and atropine.

Convalescence.—For ten days after the throat has become quite clean the child should be kept strictly at rest, lying in bed without a pillow. If all go well, at the end of ten days the child may be allowed one pillow for a week, after which two pillows are permissible. Later, the child is moved on to a sofa for increasing periods each day, and then may gradually be allowed to walk.

The size of the heart must be estimated daily. Any tendency to enlargement indicates the necessity of perfect rest.

Another daily examination that must not be omitted after the tenth day is that of the knee-jerks, for thereby warning may be obtained of an increased possibility of nervous and cardiac sequelæ. When the knee-jerks become lost the condition must be watched with the very greatest care, and as a rule absolute rest should be enforced. Cardiac symptoms of great severity may arise suddenly, so that there is the utmost need of constant vigilance in dealing with the convalescent stage of diphtheria. Anæsthesia of the soft palate is a useful early sign of post-diphtheritic paralysis.

Cod-liver oil and iron are useful in combating the anemia due to the past infection.

As the patient is not to be regarded as free from infection until no bacilli can be found in the throat, antiseptic syringing should be persevered with, three or four times a day.

WHOOPING-COUGH (*Pertussis*)

It may be well to say a preliminary word on the subject of the cough characteristic of this disease. It consists of a series of short expiratory coughing efforts succeeded by a whooping inspiration. Of the two parts, it is the paroxysmal cough which in reality is the more important, for in very young children the whooping inspiration may be present. The rapidly succeeding coughs cause the lungs to become abnormally empty, so that a violent inspiration is taken. The cause of the whoop itself is a matter of some doubt. The rush of the air may, perhaps, be so forcible as to cause the epiglottis to become folded back, thus producing a partial obstruction of the air-passage; or, more probably, there is some spasmodic closure of the glottis. The child often appears to have some warning of the approach of an attack, and will run to its mother for support. Usually the bout of coughing consists of three or four paroxysms, during which the child, leaning forward with open mouth and protruded tongue, becomes extremely cyanosed. The coughing, if at all violent, causes vomiting, and often incontinence. The attacks are said to be more frequent at night than during the day, but this statement is open to doubt. In the later stages of the disease, haler forms an important factor in the production of the paroxysm. This is shown by the fact that an attack of coughing in one child in a whooping-cough ward may cause paroxysms in many of the other children. It is common for the whoop to return several months after the cessation of the disease, should any respiratory catarrh supervene.

Etiology.—The epidemiology shows that pertussis is most common between the second and sixth years of life. Epidemics arise most frequently during the winter months, more especially after outbreaks of measles.

The incubation period is long, usually thirteen or fourteen days (seven to eighteen). Quarantine should be for a fortnight at the least. *Infectivity* lasts during the whole of the first, or catarrhal, stage, but during the actual whooping stage, provided the catarrhal symptoms have disappeared, it is certain that the child itself is no longer infectious. Six weeks will amply cover this period in most cases.

Recent evidence suggests that the disease is due to a small, aerobic Gram-negative bacillus described by Bordet.

Symptomatology.—The disease is best considered in its two stages—the catarrhal and the paroxysmal.

The *catarrhal stage* often begins with a severe cold in the head, and bronchitis. The child is a little feverish, and, though appearing well during the day, at night is fretful and off its food. Laryngitis and diarrhea are common symptoms. This stage lasts for a week or ten days, and as it progresses the cough becomes more and more paroxysmal in character, gradually causing greater cyanosis, and producing vomiting with increasing frequency. The face begins to show fullness round the eyes, which become suffused; and at length one of the paroxysms ends with a definite whoop.

The *paroxysmal stage* usually lasts for a month or six weeks. In mild cases ten or twelve attacks may occur in the twenty-four hours, but they may number up to fifty or sixty in severe cases. The child's face becomes swollen round the eyes, the injection of which increases until a very characteristic appearance is noticeable. The coughing may produce vomiting, and the vomit occasionally contains blood. A sublingual ulcer (p. 237) may be formed by the friction of the under-surface of the tongue against the teeth during the attacks of coughing; very rarely the gums may produce the same effect before the teeth are erupted. The sputum is at first very viscid, and is expectorated with the greatest difficulty. Later it becomes mucopurulent and looser. It often contains traces of blood; pertussis is, in fact, the most common cause of hæmoptysis in children. If there is much vomiting there will be considerable wasting.

Towards the end of this stage the cough becomes less violent and the characteristic whoop is heard less often, until ultimately it ceases. The presence of adenoid vegetations may tend to prolong the whooping stage.

Complications.—The most frequent are those connected with the respiratory system. The initial bronchitis may spread to the finer tubes, setting up collapse of lung and broncho-pneumonia, with all its attendant complications and dangers. These lung conditions, which are particularly liable to occur in rickety children with soft chests, constitute the great danger of whooping-cough. During any febrile complication the whooping usually ceases. Emphysema is common, but is of a mild type only. Occasionally (unusual) emphysema may develop. Acute leucorrhœa, as described by Dr. Sharkey, is sometimes originated by whooping-cough.

Hæmorrhages are not uncommon, and are usually seen beneath the conjunctiva (Fig. 48). Epistaxis is also frequent. Serious hæmorrhages are of the utmost rarity.

Nervous signs require special mention. Cerebral hæmorrhage is extremely rare. Meningeal hæmorrhage, so often mentioned in text-books, is so rare as to be almost outside the range of practical medicine. Of much greater importance are convulsions, and the meningeal

signs of rigidity and head-contraction. These may occur suddenly after a severe fit of coughing, and, with the irritation produced by constant vomiting, constitute after the respiratory complications the great source of danger in pertussis. Their cause is obscure, but they



FIG. 11. Pertussis.—Rigidity and head-contraction.

are associated with meningeal edema, and possibly with paralytic distension of the right heart consequent upon the paroxysm of coughing. Post mortem the right ventricle is often found dilated, with its wall thinned, and the brain, which is congested, shows well-marked edema. Although recovery is general, cases—more especially in wasted children—showing no pulmonary affection, and apparently doing well, may die from these nervous conditions. When at all frequent they are of most serious import.



FIG. 12. Emaciated Child Recovering from Pertussis.

Sequelæ.—The tendency of pertussis to start a tuberculous infection or to light up some quiescent focus of tuberculosis is well recognized. Occasionally, epilepsy may date from an attack of whooping-cough. The funnel-chest (fig. 40) and other thoracic deformities may follow pertussis.

Diagnosis.—During its initial stages this disease cannot be recognized unless the child is known to have been exposed to the possibility of infection.

As the cough becomes more paroxysmal in character, accompanied by vomiting and by increasing congestion of the face, the diagnosis

can usually be made without difficulty, and is confirmed by the development of the whooping inspiration. Where whooping-cough is suspected, the examination of the throat by means of a spatula may bring on a characteristic attack. It must be remembered that infants under one year of age often do not whoop at all during the illness.

Enlarged tuberculous mediastinal glands may produce a similar cough, but without the whooping inspiration. In such cases, however, the signs of tuberculous are usually patent. The progress of the case will decide the matter. Some cases of empyema show a paroxysmal cough resembling that of pertussis, but ending without any whoop. The crow of laryngismus stridulus somewhat resembles as sound the whoop of pertussis, but the history of the case is so different that no real difficulty arises from the similarity.

Course and Prognosis.—In an uncomplicated case in a favourable subject the disease runs a course of about eight to ten weeks from start to finish. In the cold weather the whooping stage may be prolonged, and may not entirely disappear until the advent of a warmer season. Adenoids, again, may keep up the paroxysmal cough. Mention has already been made of the possible return of the characteristic cough, even after several months. In neurotic children the disease tends to run a severe and prolonged course.

In infants pertussis is always a matter of anxiety, owing to the possibility of pulmonary collapse. Rickets, too, which tends to the production of pulmonary complications and convulsions, makes the outlook more grave. The majority of deaths from this disease occur in children under the age of three years.

Treatment.—As we know of no drug by which the course of the disease can be shortened, it is well to remind ourselves of the object of our treatment. That object is twofold:—to guard against, or to treat, pulmonary complications, and to diminish the violence of the cough, upon which depend both the running (and so the general nutrition of the child) and the tendency to sybotic catarrhitis.

The child should be kept in bed during the catarrhal stage, or if the temperature is raised. Later, during the paroxysmal stage, if there are no complications, it is better that the patient should be allowed up and out in the open air if the weather be mild. Games should be permitted, but care must be taken that the child does not become unduly excited. The room should be well ventilated, and a good supply of fresh air ensured. When possible the child should be given two rooms, one for the day and the other for the night. It is a common practice to charge the air of the room with some vapour, such as eucalypti, Pine's balsam, or carbolic acid, but the remedial effect of such a procedure is, at the best, very doubtful, and should it lead, as it too often the case, to the apartment being kept close and stuffy, it is better omitted. At the same time every precaution must

be taken to prevent the child being exposed to such conditions as will lead towards bronchitis or pneumonia.

The food should be light, nourishing and easily digested and should be given in small quantities at frequent intervals. Should the cough cause vomiting, it is best to give small feeds directly after the paroxysms.

During the catarrhal stage the bronchitis should be treated according to the lines laid down under the treatment of that condition (p. 149). Expectorants may be of distinct service during this part of the illness. Where the sputum is very viscid and expectorated with great difficulty, alkalies may be of use.

To control the violence of the cough various drugs may be used. Of these belladonna, antipyrin, bromide, chloral hydrate, bromoforn, alkalies, opium, and opiodol acid are perhaps the most useful. Belladonna, which may be given with alkalies or potassium bromide, should be pushed if any good effect is to be produced. To start with, six minims of the tincture may be given to a child of three years, and should the cough remain uncontrolled the dose may be gradually increased to ten or twelve drops every four hours. Slight flushing of the skin is perhaps the best sign of the proper dose having been administered; dilatation of the pupils and dryness of the mouth are to be avoided. Even in small infants large doses are well tolerated. For an infant of one year old the following is a common mixture. The laevicase may be replaced by some sodium bicarbonate if desired—

R.,	Tinct. Belladen.	Rij	Syrup. Tolu	Rjss
	Ext. Froes.	gr. ij	Aq. Distill.	ad 5j
	Ammon. Carb.	gr. 4		

In older children, antipyrin may be given in 1- to 3-grain doses three times a day; in the younger ones a grain for each year may be given. Bromide and chloral hydrate are useful where there is a tendency to convulsions.

Should convulsions arise following the cough, oxygen should be administered immediately, and strychnine given hypodermically. If the child is very ill, immediate lumbar puncture should be performed and a few drachms of cerebrospinal fluid withdrawn. If necessary, artificial respiration may be resorted to. Venesection is occasionally of great service. Should the cough be very severe, it is well to have oxygen at hand in case it is needed in a hurry.

It considerably lessens the irritability of the cough if more efficacious than a change of air, which may cause the cough to disappear quickly. If adhesions obstruct respiration, they should be removed; their removal is sometimes followed by a cessation of the cough.

CHICKEN-POX (*Varicella*)

First separated from variola by Hebra in 1767; varicella gains

its chief interest from the close similarity which may exist clinically between it in its severest forms and modified small-pox.

Etiology.—Neither the age nor the seasonal incidence show any striking peculiarity. It is perhaps most common in the autumn months, and in children of three or four years old. It has been reported in an infant on the second day of life (Hibbard). The incubation period is from ten to nineteen days, usually from thirteen to fifteen; a quarantine free of twenty days is therefore necessary. A second attack of chicken-pox is of the utmost rarity, the immunity afforded by this infection being more complete than is the case in any other specific fever, with the possible exception of whooping-cough. Variella may co-exist with whooping-cough, scarlatina, or measles.

Symptomatology.—The symptoms of invasion may be entirely absent and the rash may be the first sign of illness. In many cases, however, there are slight constitutional symptoms, consisting of a mild degree of pyrexia with some headache or irritability. In children these are rarely severe, and do not precede the rash by more than twenty-four hours. Rarely there is at this time a profusional scarlatiniform rash on the skin of the trunk, and there may be slight conjunctival injection.

The eruption usually appears first on the trunk; the face and scalp not uncommonly, and the limbs very rarely, showing the first lesions. The rash appears in crops lasting over a period of from three to five days, occasionally longer, and the temperature may be slightly raised with the eruption of each fresh crop. In addition to the sites named, the mucous membrane on the buccal surface of the cheeks, the palate, gums, or tongue may become involved, showing cracks which rapidly rupture and give rise to areas of softened epithelium or of ulceration. The conjunctiva and the mucous surfaces of the genitals are less commonly affected. The palms and soles are but rarely affected, and even should they be the rash does not go beyond the papular stage. The number of poeks is directly proportional to the severity of the infection. However thick the rash, it is never confluent.

The earliest lesion which may be present is a small red papule. Should it appear it is only visible for a few hours, and this stage is frequently absent altogether, the vesicular being the first seen. The vesicles are large, round, or oval in shape, clear and unilocular, so that when pricked they may be easily emptied by pressure. They are surrounded by an area of inflammation of varying depth. They appear superficial, and are not indurated at their bases except where they occur on the forehead. They are dome-shaped and not umbilicated, but may become so when shrinking or if ruptured by scratching. Lasting for about forty-eight hours, they then become opalescent, and begin to shrivel, leaving behind hard scabs which take a week or ten days to separate. On the fourth day of the disease the

rash can usually be seen in all its stages. It is very irritating, and the affection of the groin may give rise to a considerable amount of discomfort. When the vesicles have suppurated as the result of scratching, a permanent white scar may remain.

Variations.—Three main types of chicken-pox may be mentioned briefly. *Varicella bullosa* is the name given to a variety in which the vesicles are so large as to form bullae. Such a condition may closely resemble pemphigus, but a few vesicles of the ordinary type are present. In other cases (*varicella gangrenosa*) the bases of the vesicles may become gangrenous and lead to sloughing, which may be extensive. The temperature remains raised and, unless only a few of the poeks are affected, the case usually ends fatally. In *varicella hæmorrhagica*, bleeding may occur into the skin at the base of the poeks, and even from some of the mucous membranes.

Diagnosis.—The more severe attacks of chicken-pox may be easily confused with small-pox in its modified forms, and serious mistakes have arisen in the past. So difficult is it to distinguish, that usually *varicella* is made noticeable during an epidemic of small-pox. The chief points of difference must be mentioned, but each one is subject to variation. The symptoms of invasion in *varicella* tend to be mild, and to last for not longer than twenty-four hours. The variety with the rash delayed until the third day probably does not occur in children. In *variola* this stage is usually associated with much prostration, pain, and collapse, and lasts for three days. The rash in *variola* usually appears all at once, and shows well-marked vesicular and papular stages, while in chicken-pox the rash comes on in crops, and the papular stage is either absent or very transient, so that the full vesicular stage is very quickly reached. The vesicle in chicken-pox is larger, more superficial, and with less induration round its base than that of small-pox. It is also dome-shaped, not flattened nor umbilicated as in *variola*. The presence of scars due to past *varicella* almost precludes the possibility of a present infection being that disease. The scars due to *variola* are rarely seen in people who have been well vaccinated, and if present they are more abundant on the face than on the trunk, whereas the reverse obtains in the scarring due to chicken-pox. Lastly, the temperature in *variola* falls rapidly when the rash develops, and does not rise again until suppuration occurs. In *varicella* the temperature tends to be raised, though usually only slightly, during the development of the various stages of the eruption.

Treatment.—The patient must remain isolated until the scabs have separated. If the groin is painful, the diet should be soft and constipatory. The child should be prevented from scratching the poeks, especially those on the face. This may be effected by keeping the arms extended in light spirits if necessary. The itching may be

very severe, and, should it cause sleeplessness, may be temporarily relieved by warm sponging or by a warm bath. Carbolic oil or boracic-acid baths may be used. In the gangrenous variety, supporting and stimulating treatment will be necessary, and to the affected parts compressions or some suitable dressing should be applied.

MUMPS (*Specific Parotitis*).

Etiology.—Specific parotitis or mumps, as opposed to a diffuse inflammation of the parotid gland (p. 255), is an infectious disease which occurs in epidemics. Most common in children of from five to fifteen years, and during the winter and spring months it is usually spread directly from the patient to the healthy, though probably it may also be conveyed by an intermediate person. It has a long incubation period of three weeks (fourteen to twenty-four days). The child should be regarded as infectious for four weeks from the onset of the illness, provided that all swelling of the affected parts has disappeared for a week. Quarantine, when necessary, should be for twenty-four days. A second infection is very rare.

Symptomatology.—The disease usually starts with pain behind the angle of the jaw, which is aggravated by opening the mouth, and with swelling of the parotid gland, which quickly appears. Associated with the pain and swelling, some pallor, rashiness, and a slight rise of the temperature occur. These symptoms may precede the local ones by a day or two. Occasionally the constitutional symptoms are of greater severity, the temperature may rise to 101° and there may be some delirium at night. A good deal of pain is caused by mastication owing to the increased flow of saliva which accompanies this process. The tongue may become brown and the mouth dry, since the child breathes with its mouth open. The skin over the parotid is occasionally reddened. Often the swelling spreads to the submaxillary glands, and on rare occasions the parotid glands themselves escape infection. Although both sides of the face may be affected at once, it is more common for one to become swollen and tender three or four days after the other, the temperature being again raised. The swelling of the affected glands usually lasts for five or six days.

A form of the disease has been described under the name of malignant mumps, in which there are very high fever, unconsciousness, delirium, and rigidity, with head-retraction without meningitis. The swelling extends down the neck from the parotid, and is hard, red, and tender; the tonsils are enlarged, covered with exudate, and ulcerated. Death occurs within a week. The exact relationship of this condition to true mumps is perhaps rather doubtful.

Complications.—These are very uncommon. Orchitis has been reported in a boy of twelve years, but is infinitely rarer under that age. The homologous affections of ovaritis and mastitis hardly ever occur in

Infant children under twelve years of age. Suppurative parotitis may arise, as from any other condition in which the mouth is abnormally dry, but it is very uncommon during mumps. Monilia and other media have likewise been recorded, but are probably associated with a septic infection of the parotid.

Diagnosis.—Enlargement of the lymphatic glands from pediculosis capitis, conjunctivitis, swollen teeth or facial diphtheria may give rise to difficulty. The parotid gland, however, in these conditions is not enlarged, and there is not the characteristic pain in opening the mouth. The condition of the socia parotides, which is swollen in mumps, should be examined. Septic parotitis causes swelling of the parotid gland, but is associated with some condition in which a very dry mouth has been present. It often results in suppuration.

Prognosis is invariably good. Some chronic enlargement of the gland may persist after the attack, but it usually clears up in time.

Treatment.—The mouth should be sprayed frequently with some antiseptic lotion such as that of perchloride of mercury (1-2,000) in order to keep it moist and clean. For the pain, fermentations of glycerin and belladonna may be used and, if necessary, a little Dover's powder may be administered. The diet should be such as does not require mastication, milk, custards, jellies, etc. Should there be much delirium, warm baths or sponging may be ordered.

As a rule a child with mumps will need to be kept in bed for about seven days.

SECTION I

DISEASES OF THE DIGESTIVE SYSTEM.

I.—DISEASES OF THE MOUTH.

STOMATITIS.

Clinical Varieties.—Several varieties of stomatitis are found.

Catarrhal Stomatitis.—This is very commonly seen in infants during the dentition period. It is usually associated with some catarrh of the respiratory tract or of the stomach and intestine. Eruption of the teeth is painful in the presence of stomatitis. Small areas of the mucous membrane of the mouth become reddened and their surface granular or covered by a thin white film of swollen and disintegrating epithelium. Salivation is present.

Aphthous Stomatitis.—This is a common condition occurring with greatest frequency in children between the ages of one and three years. It is probably of an infective nature. The aphthæ are small, greyish-yellow areas, surrounded by a zone of hyperæmic mucous membrane. They occur on the tongue, lips, and gums, and may spread to the cheeks or palate.

Ulcerative Stomatitis.—This occurs in infants, particularly in the ill-nourished. It may be associated with scrofulic signs in the germ. It is also seen in older children, and generally affects those of poor physique. The appearance of the mouth is similar to that in the aphthous variety, but here ulceration, superficial or deep, is present. The saliva may be blood-stained. Very rarely in rooted children the ulcerative changes may lead to gangrene (*necrosis oris*).

Parasitic Stomatitis (Thrush).—Thrush is due to a yeast fungus, the *oidium albicans*. It is found chiefly in ill-cared-for infants with gastro-intestinal disorders. The amount of stomatitis is very variable, and patches of thrush may be found without any non-parasitic stomatitis. This, when present, may be the primary or secondary condition. Thrush appears in the mouth as small white patches which resemble pieces of curdled milk, but which do not disappear on being gently sponged. It is found upon the tongue, cheeks, and fauces, and may extend down the œsophagus into the stomach. Frequently in infants with thrush an erythematous rash or a dermatitis appears on the buttocks, popularly supposed to be due to the thrush "going through" the child. The fungus itself, however, rarely causes this condition, which may be due to the acid stools of the associated diarrhoea, or to syphilis.

Gangrenous Stomatitis (Cancerous).—This is best considered separately from the other forms of stomatitis (p. 257).

Symptomatology.—As the result of the soreness of the mouth refusal to take food is a frequent symptom, and stomatitis is one of the conditions which have to be considered when a baby suddenly refuses its bottle. Dentition, should such be occurring, is rendered painful. In older children teeth may become loosened. Salivation is almost constant. In the ulcerative variety the saliva may be blood-stained. In the milder types of the disease there is little constitutional disturbance, but in the severer forms there are pallor, fever and wasting. In some cases circum-oral pallor is very marked.

The tongue is generally coated, and in infants there are usually symptoms due to involvement of the rest of the alimentary tract, such as vomiting and diarrhoea, of which the latter is the more constant. Periculis may also be present. In older children these symptoms are not so marked, and loss of appetite and constipation are more seen. The disease lasts as a rule about a week.

Treatment.—Attention must first be paid to the treatment of the gastro-intestinal tract, and in many cases no local treatment is required. In infants a dose of castor oil may be given, followed, if necessary, by the regular administration of a castor-oil mixture. In older children calomel is advantageous, followed by calomel and soda powders, or by a mixture of rhubarb and soda. Scurvy if present, must be treated.

Scrupulous cleanliness in connection with all feeding utensils should be observed.

For the mouth various local applications may be used. A simple alkaline solution (5 grains of soda to the ounce), glycerin of borax and the honey or glycerin of borax and many other solutions are of use. Solol, dissolved in a little alcohol and mixed with glycerin, acts well. In older children lozenges containing formaldehyde may be ordered to be dissolved in the mouth. They are pleasant, and children usually like them. Potassium chlorate given internally is perhaps the most valuable drug in remedying the condition of the mouth. For an infant of one year old such a mixture as the following may be prescribed:—

R. Pot. Chlorate	gr. iiss	Syrup	℥ss
Inf. Fern Ferchard.	℥iss	Aquam.	ad 3i

In the severest cases the child should be put to bed and the mouth washed out with solutions of potassium chlorate, persulphate of mercury, Eschsch, or hydrogen peroxide. Chlorate of potash should be given internally.

During convalescence, when the digestion is normal, tonics and iron may be necessary. The teeth in some cases require a dentist's care.

CANCERUM ORIS*(Gangrenous Stomatitis).*

This is practically confined to anemic and ill-nourished children. It is more common in girls than in boys. It may occur at any age, but is commonest between the ages of two and five years. It most frequently develops during or after measles, but any acute infection may precede it.

Symptomatology.—Beginning as a red, irritated patch on the inner side of the cheek, its edges rapidly spread while the central part becomes dark and gangrenous. In its earliest stages the condition is apt to be overlooked, and a local swelling on the outside of the cheek may be the first sign of cancerum oris recognized. Drifting of the saliva is present, and the discharge is very offensive. The child rapidly becomes severely prostrated and often unconscious. The gangrenous ulceration may extend until the cheek becomes perforated.

Prognosis.—In the majority of cases the disease is fatal. Death may occur from anemia, septic broncho-pneumonia or pyæmia. Occasionally the process becomes stationary and healing occurs with consequent deformity of the face.

Treatment.—This is very unsatisfactory. Every attempt should be made to prevent the onset of septic broncho-pneumonia, but it may be that in some cases it is present from the first. The child should be laid upon its side, so that the saliva may drain from the mouth. Where the lungs are apparently free from disease an early tracheostomy has much to recommend it.

The ulceration in the mouth should be thoroughly cauterized and painted with carbolic acid or cauterized with nitric acid. Perchloride of mercury may be used as a local antiseptic. Where perforation of the cheek has occurred, the sloughing edges should be trimmed surgically.

SYPHILITIC AFFECTIONS

of the mouth, tongue and fauces are considered in the section on **TOXICITUM SYPHILIS** (p. 185).

SUBLINGUAL ULCERATION.

This is a condition of minor importance. It consists of a small, clean-cut ulcer on each side of the freedom of the tongue, and is due to the constant local injury done by the lower incisor teeth in cases of violent cough. It is extremely rare where no teeth have been cut. It is seen in cases of pneumonia, but more commonly in those of whooping-cough. Even here, however, it is of no diagnostic value, as it is not usually present until definite whooping has occurred.

Treatment.—The mouth should be kept clean. Local treatment such as has been detailed under stomatitis is orders required, and it is of use until the violence of the cough has abated.

DENTAL CARIES.

This is an extremely common disorder of childhood. According to Dr. Still, 82.7 per cent of the children of the hospital clinics between the ages of five and twelve years, and 39.8 per cent between the ages of two and five years have carious teeth. They are hardly less common in children of the better classes.

Etiology.—Rickets and malnutrition predispose towards soft caries by causing a poor development of the enamel covering of the teeth. The changes of decay are due to many factors, of which may be mentioned over-crowding and irregularity of the teeth, lack of cleanliness, excess of carbohydrate in the diet, the eating of cakes and sweets between meals, and gastric indigestion.

Symptomatology.—To the doctor the hospital mother rarely mentions the word toothache, but complains rather that the child refuses its food or, what is the same thing to her, teeds a fever. Some painful dental condition has to be remembered as a possible cause of "loss of appetite." On the other hand, dental caries may be responsible for headaches and swollen glands, or for indigestion, which arises in part from the swallowing of septic material and in part from the holding of food which may ensue where the teeth are tender. Habit-spasms, and even epilepsy, on rare occasions, may be set up by carious teeth.

Treatment.—Dental caries in children must not be neglected. It should be prevented as far as possible by attention to the diet, by cleanliness, and by regular visits to the dentist. Where present, it must be treated in children as in adults. The mouth must be cleansed by mouth-washes and by the use of the toothbrush. Where possible the teeth, even first teeth, should be stopped; but where this is impracticable they should be extracted.

II.—DISEASES OF THE SALIVARY GLANDS.

PAROTITIS.

Specific Parotitis (Mumps).—This is considered on page 252.

Septic Parotitis.—This occasionally arises in children after scarlatina, measles, and other infections. It occurs where the mouth is allowed to become dry, as after abdominal operations or during the

administration of belladonna. Facial palsy may on rare occasions be caused by this condition.

Treatment.—In the early stages the mouth must be kept both clean and moist, and fomentations should be applied to the swollen gland. Later, should suppuration occur, it may be necessary to open the abscess from outside.

III.—DISEASES OF THE OESOPHAGUS.

From a practical point of view, the fact best worth remembering about abnormal conditions of the oesophagus is that a foreign body, such as a coin, lodged here below the larynx, may give symptoms of obstructed breathing with little or no dysphagia.

Both thrush and diptheria may spread down the oesophagus into the stomach. Perforation of the oesophagus by a cancerous tubercular gland is a rare event, and seldom gives rise to any immediate symptoms. Acute oesophagitis may be produced by scalding, and occasionally gives rise later to scars of stricture. Slight bruising of the oesophageal wall from the repeated passage of oesophageal tubes may occur at the level of the larynx. It does not appear to do any harm. Hemorrhage from this cause is very rare. Some dilatation of the last inch of the oesophagus, with hypertrophy of the musculature, is seen in many cases of infantile hypertrophic pyloric stenosis.

Congenital malformations are extremely uncommon. Diverticula may exist, but rarely give rise to symptoms during childhood. The upper end of the oesophagus may open into the trachea, the larynx terminating blindly. Rarest of all, the lower end of the oesophagus may fail to communicate with the stomach.

IV.—DISEASES OF THE STOMACH.

VOMITING.

So many are the causes of vomiting in infancy and childhood, that not more than a rough classification of them can be attempted here.

In infancy vomiting is so frequently present that we must be able to recognize whether it be of serious moment or not. The only guide in this matter is to be found in the child's progress as estimated by the gain of weight. Where this is satisfactory, the vomiting may be regarded as due merely to overfilling of the stomach. Such "overflow vomiting" usually occurs painlessly soon after a meal, a little unchanged milk being possetted up with, perhaps, the eructation of some H_2 which has been swallowed. Beyond reducing the volume of the food given at each feed, no treatment for this is necessary. On the other hand, where the vomiting is associated with failure to gain

tough, it is a symptom which requires careful attention. In such a case there is probably something wrong with the feeding or with the child's stomach. The food may be of an unsuitable nature, too large in volume or too quickly administered, or the feeding may have been given at irregular intervals. The abnormal conditions of the stomach which give rise to vomiting and scawing are atrophic dyspepsia (in marasmus), and dyspepsia (pyloric spasm) and infantile hypertrophic pyloric stenosis. These require careful differentiation (p. 272). Large vomits in infants may cause serious, even fatal collapse.

Many abdominal disorders are associated with vomiting. In infants both diarrhea and constipation are usually accompanied by it. Should serious vomiting be present from birth, congenital stenosis of the intestine, usually of the duodenum, may be suspected. Various forms of intestinal obstruction and of disease of the peritoneum account for a large group of cases in which vomiting is a permanent symptom.

In diseases of the respiratory system vomiting may be due to the violence of the cough, and is most frequently found in whooping cough. In pulmonary tuberculosis, in addition to this cause, it may be due to swallowed sputum, peritoneal adhesions, or meningitis.

In heart-disease, either primary or secondary to pulmonary disease, vomiting is a symptom of right heart failure.

In infections, vomiting is frequent at the onset of the disease, and probably takes the place of the rigor in an adult. It is almost constant at the onset of scarlatina, and very common in primary pneumonia.

Vomiting is often due to nervous causes. Some infants will induce it by cramping their fingers into their mouths. Others acquire the habit of regurgitating their food into the mouth, such rumination being evoked by movements resembling those of swallowing. In some older children it may be due to distaste for a certain kind of food. Sometimes a child will vomit solid food because it dislikes it, while digesting a fluid diet perfectly. In less degree such a condition is very common in neurotic or spoiled children, who will vomit successfully all forms of food which they dislike (such as rice pudding), while tolerating amazing quantities of such dainties as chocolate with pink inside."

In organic nervous diseases vomiting is usually due to raised intracranial pressure, and occurs in such conditions as meningitis and intracranial tumour or abscess. Typically such vomiting is sudden and explosive and unaccompanied by nausea.

Lastly, vomiting may conform to the type known as recurrent cyclical vomiting, or be associated with other conditions in which acid intoxication is present (p. 362).

INFANTILE HYPERTROPHIC PYLORIC STENOSIS.

This disease has gradually become recognized as one of no uncommon occurrence. Indeed, so frequently is it seen that so one can now

afford to be unfamiliar with it. Moreover, the prognosis in it depends to a large extent upon its early recognition. For these reasons a somewhat full account of the disease is necessary.

Nomenclature.—As in the majority of cases at birth there are no symptoms of stenosis of the pylorus, it seems more accurate to use the term "infantile" rather than "congenital" in the title of the disease.

Etiology.—While much remains obscure as to the actual cause of the condition, there are some clinical points of importance to be mentioned under this heading. The disease is from eight to ten times more common in boys than in girls. It may occur anywhere in a family, but about one-third of the cases arise in firstborn children. I have seen it in an eleventh child. It is excessively rare for more than one in a family to have it, although such an occurrence has been reported. I have seen one of twins affected and the other normal. The nature of the labor and the terms of pregnancy seem of no significance. About an equal number of cases, in my experience, arise in babies given bottle, breast and mixed feeding.

The theoretical considerations concerning the causation of the disease may be left until its needed anatomy has been described.

Morbid Anatomy.—The changes found in the stomach are usually quite distinctive. The pylorus is enlarged in its circumference, and to the touch feels of almost cartilaginous hardness. The wall of the stomach is hypertrophied, as can easily be recognized by palpation. Owing to this thickening it is pearly-white in colour. The size of the stomach is very variable, depending to a large extent on whether death has occurred when the organ was in systole or diastole. If the former, the stomach is not enlarged but has a remarkably substantial appearance, and its walls show the pearly-whiteness well. If diastolic, the organ is greatly enlarged, and may occupy the major part of the upper abdomen; owing to their comparative thinness, the walls are not so striking in their appearance as in the contracted organ, but their hypertrophy is easily recognizable on palpation. The pylorus itself lies to the right of the vertebral column 23 or slightly above the level of the lower border of the liver. It is important to notice that when the stomach is enlarged the pylorus is tucked away behind the pyloric end of the organ (Fig. 50).

When the stomach is opened, the thickening of its walls is seen to increase in proportion as the pylorus is neared. The hypertrophy commonly extends to the last inch of the oesophagus. The pyloric wall itself is greatly thickened, measuring between an eighth and a quarter of an inch. The increase is practically confined to the circular muscular coat, which forms a hard fleshy mass. The thickening develops gradually on the stomach side, but comes to an abrupt end on the duodenal side (Fig. 51). The pylorus after death easily allows of the passage of a small quill.

The mucous membrane very constantly shows signs of gastritis and a rather reddened and injected, or grey and swollen, in appearance, and is covered by a variable amount of mucus. In the pylorus the mucosa is thrown into longitudinal folds, usually three in number. It is to be noted that the pyloric mucus, as seen at operations, appears very congested and oedematous.

The condition of the small intestine is important. It may be contracted and atrophic to a most remarkable degree, but whether empty or distended with air, its walls post mortem always show marked wasting.

Associated malformations, which have been reported in a certain number of cases, require mention as their occasional presence has been brought forward to support the theory that the disease is due to a congenital malformation. Such conditions as slight ectopia



Fig. 10.—Hypertrophic pyloric stenosis.
Kilnschmidt's post mortem. Note pyloric sphincter and coiled portion of duodenum.

of the toes, slight dilatation of the renal pelvis, as well as congenital cardiac lesions, cerebral and other cysts, have been reported; but even where all minor abnormalities are included, the percentage of the cases with malformations is too small to be of any weight in the argument on the causation of the disease. The *only* malformation which is at all common is ptosis. This is nearly constant, and probably accounts for the dilatation of the pelvis of the kidneys, which has been reported.

Theoretical Considerations.—Of the theories which have been advanced to explain the origin of hypertrophic pyloric stenosis, two only need be considered. In the first it is supposed that at the time of the child's birth the pyloric wall is thicker than normal, and that

from this there arises, either immediately or later, stenosis of the outlet to the stomach. This theory, then, assumes the presence of a congenital anatomical abnormality. The second theory is to the effect that there is no such malformation, but that spasm of the pylorus, set up in the early days of life, and occasionally before birth, accounts for the hypertrophy and for the stenosis. In both views the hypertrophy of the stomach is compensatory in order to overcome the stenosis at the pylorus.

These two views require some consideration, for various authorities—unfortunately as it seems to me—have very dogmatically laid down



Fig. 10.—HYPERTROPHIC PYLORIC STENOSIS.

Stomach showing hypertrophy of pyloric and gastric muscles, also filling of pyloric antrum.

rules for the treatment of the disease founded upon their speculations as to its origin.

That the disease is due to a congenital malformation is supported to some extent by its sex-incidence and its frequency in firstborn children. It is difficult to see why an acquired disease should show these two peculiarities, while such might occur in a congenital malformation. The view is also supported by the fact that a hypertrophied pylorus has also been found in a fetus (Dent). Against this theory, however, are several important points. That the circular muscular coat of the pylorus should alone be involved is without question peculiar. That the symptoms of stenosis, usually

absent at birth, should come on later, is difficult to explain by the theory alone: for a gradual narrowing of the pyloric opening is unlikely to occur when the abnormal condition of the wall is due only to excess of muscular tissue, there being in these cases no evidence of hypertrophy or inflammation of the pyloric wall such as might cause consequent stricture. Further, it is quite beyond question that many cases have completely and permanently recovered without operative treatment, a fact which is difficult to explain if the condition be due to an organic stricture. Lastly, in a case recorded by Dr. F. L. Batten, recovery had taken place six months previous to an attack of pneumonia which proved fatal, and post mortem the pylorus was found much hypertrophied, although it was evident on clinical grounds that no stenosis had lately been present.

On the theory that the pyloric hypertrophy is due entirely to spasm, it is very difficult to account for such cases as do show symptoms of stenosis at birth. Nor do we understand why such severe spasm should be set up in the pylorus, either before or after birth. Hypertrophic pyloric stenosis has nothing to do with acid-dyspepsia, as Dr. Wilcox and I were able to show, the two groups of cases standing separately, and apparently unconnected by intermediate cases. That only the circular coat of the musculature of the pylorus is involved is in favour of this theory. In young children hypertrophy is very readily induced by over-action, and it might well be that severe spasm, which is of course quite a different thing from a constant tonic contraction, could originate hypertrophy. Lastly, the occurrence in the course of disease of periods in which symptoms of stenosis are absent is in favour of spasm playing an important rôle in the condition.

It is seen, then, that there is much to say for and against each theory. Certainly we cannot hold on such disputable theoretical grounds, that operative treatment is always necessary or always unjustifiable. The apogee of the argument is, not to put too fine a point upon it, that we do not yet know the cause of the disease.

But there is a further point, and one of much greater practical importance. While the origin of the disease remains obscure, the origin of its symptoms is extremely plain. The initial symptoms certainly arise from the difficulty of getting food through the pylorus. Now this difficulty can be due to three things only: firstly, an organic stricture of the pylorus; secondly, spasmodic closure of the pylorus; and thirdly, gastritis, which acts by causing swelling of the mucosa of the pylorus, thus tending to occlude the lumen, and by entangling the food in mucous mashing it from bulky and less capable of passing out of the stomach.

The practical application of this point is quite simple. Treat the spasm and gastritis by lavage and dieting, and if food still cannot be passed through the pylorus, operative interference will be necessary. In practice we find that by lavage and careful feeding, in the course of a week and often less, food passes through the pylorus in quantities

are only sufficient for the patient's needs, but only too often sufficient to set up severe diarrhea in the very atrophic intestines. If at the end of a week or fortnight there is still occlusion at the pylorus, operation is indicated. Where, however, medical treatment has been properly carried out, the pylorus almost invariably permits the passage of food satisfactorily. From this we see that the prognosis of the disease depends to a very great extent, not upon the condition of the pylorus, which is amenable to treatment, but upon the assimilative power of the intestines, which may be, and very commonly is, so deficient as to render a fatal issue inevitable. To these matters further reference will be made under the treatment of the disease.

Symptomatology.—The three prominent symptoms are vomiting, constipation, and wasting. In about 70 per cent of the cases these start during the second and third weeks of life, but they may be present at birth or may be delayed as late as the twelfth week. Not uncommonly the vomiting is said to start at birth, but on careful enquiry it is found that it was not abnormal nor accompanied by constipation and wasting until a later date. At the onset the vomiting occurs two or three times a day, but as the case proceeds it becomes more frequent, until it follows each feed and is more projectile and more bulky. In some of these cases the vomiting is so forcible that the contents of the stomach are shot two or three feet across the bed. Large vomits are frequent, and may cause serious or fatal symptoms of collapse. Vomiting is in some instances absent for several consecutive days even in intussuscepted cases. The vomitus consists of curdled milk in almost colourless fluid, and contains large stringy pieces of *clara* mucus. Streaks of blood are occasionally present, while small clots of blood are very rare. The tongue is usually coated. Abdominal pain is not a persistent symptom of the disease.

With serious vomiting the constipation becomes very severe. Small dry pellets of fecal material are passed, the bowels acting perhaps every two or three days. In most cases the wasting rapidly becomes extreme. The skin is tightly stretched over the bones of the face (Fig. 52). The faces of the patient, with its prominent features, becomes rather



Fig. 52.—Hypertrophic Pyloric Stenosis.
(Facing thumbing.) (From *Transactions Pyloric Stenosis* (part I).)

characteristic; for such severe emaciation is seldom seen in very young infants apart from this condition, although very obstinate constipation of a single type sometimes produces a similar appearance.

The gastric contents on analysis show definite changes, as Dr. Wilkos and I were able to demonstrate.* If a small test-meal of milk and water be given, and the stomach emptied after the lapse of half an hour, a large yield is obtained, showing that there is retention of food in the stomach. Mucin is present. The ferment activity (estimated as remains) is much increased, while the total acidity is diminished. The excess of mucin and the lessened acidity are evidences of the presence of gastritis, while the increased ferment activity is due to the hypertrophic state of the gastric walls, and in very early cases may not be present.

While these are the usual symptoms, the common dangers of the condition must be mentioned. One of the most important is a fatal syncope attack. This occurs suddenly, and may be quite unavoidable, but more often it immediately follows a large vomit. So great is this danger that it must be kept in mind throughout the disease, and distention of the stomach with retained food must not be allowed to occur. Diarrhoea is another symptom which accounts for many deaths in this disease. Owing to its atrophic condition, the intestine can deal with only very small quantities of food, and any excess quickly sets up diarrhea. The termination of the case may simulate that of simple marasmus with hyperpyrexia, rigidity, and heat-retraction. The last symptom is very occasionally due to a neuritic thrombosis of the superior longitudinal sinus.

Nearly the whole art of treating this disease lies in the avoidance of these two dangers, diarrhea and large vomits which cause collapse.

Physical Signs.—These are two in number, and are quite distinctive of the disease. They consist of visible gastric peristalsis and a palpable pyloric ferment.

The peristalsis is best seen after a feed. Characteristically, a swelling appears from under the left costal margin, passes slowly across the abdomen to the right of the umbilicus, and disappears. Usually before the first has subsided a fresh one makes its appearance, and often three swellings may be seen travelling from left to right under the abdominal wall (Fig. 33). Where the dilatation of the stomach is great, the waves may be seen to pass over into the right flank. Peristalsis is best provoked by administering a feed, but may sometimes be set up by stroking the abdominal wall with the finger. It is to be noted that in some cases no peristaltic waves are seen until the patient has been under treatment for two or three days. In such cases a thickened and contracted stomach may be visible and palpable under the abdominal wall.

* *Lancet*, Dec. 14, 1907.

The pyloric tumour is best felt while peristalsis is proceeding, as during this the stomach is straightened and the pylorus uncovered. At other times the pylorus lies close to the vertebral column, tucked away behind the body of the stomach (*Fig. 50*). For this reason it is necessary that the palpating hand should be placed well down in the right flank, pressure being made in an upward and inward direction towards the spine. Unless this be done, the thickened gastric walls which lie over the pylorus will prevent the recognition of the



Fig. 50.—HYPERTROPHIC PYLORIC STENOSIS.

Showing gastric peristalsis. In the upper two pictures the irregular outline of the stomach is seen; in the lower typical peristaltic waves passing across from the right flank towards the spine.

tumour. The pylorus feels like an enlarged gland, hard but freely movable, lying slightly above the level of the lower border of the liver, by the side of the spine.

Examination for the pyloric tumour is not sufficient to promote vomiting. Where then this sign has once been found to be definitely positive, it is useless, if the child's condition be serious, to endeavour to elicit it again.

Diagnosis.—The symptoms of this disease—those of vomiting, constipation, and wasting—may be simulated in various conditions in very young children. Firstly, in cases of severe constipation, emaciation may be extreme, but by the comparative softness of the gastric symptoms, and the absence of peristalsis and pyloric tumour, this group is usually easily differentiated. In the second class, that of acid dyspepsia of infants, or pyloric spasm without hypertrophy, much difficulty may arise in the matter of diagnosis. The vomiting, constipation, and wasting are all present, and the vomits may be large, frequent, and even projected. These cases can be differentiated by the following facts. They occur as a rule at a slightly later age, or perhaps more accurately, they come under observation later than the hypertrophic pyloric cases. They show no peculiar sex-incidence. The emaciation caused is not severe. There is much screaming from colic. The tongue is usually red and clean. The vomit contains no mucus; its ferment activity is low and its total acidity high. Lastly, there is no true gastric peristalsis, nor is there a palpable pyloric tumour (see Table 22, p. 272).

The peristaltic waves can only be simulated by one condition, namely, congenital dilatation of the colon (p. 282). In this, which is a very uncommon disease, the enlargement of the bowel may be so extreme that the sigmoid passes across the lower abdomen, and in it left-to-right peristalsis may occur. The symptoms here, however, are rectal rather than gastric. In acid dyspepsia the enlarged stomach may be seen standing out under the abdominal wall, but no peristalsis occurs in it.

The pyloric tumour, if felt, can hardly be mistaken for anything else. I have once seen a mucous cyst of the wall of the pylorus produce a tumour which, had it been felt, would have simulated that of a hypertrophied pylorus.

Mucus in the vomit, in a case of chronic vomiting in a small, wasted infant, is very suggestive of hypertrophic pyloric stenosis. A very marked increase of the ferments is apparently diagnostic of the condition. Where 10 cc. or less of the filtered gastric contents completely solidifies 5 cc. of milk at 40° C., the diagnosis is probably certain. Absence of increased ferment activity does not, however, preclude the possibility of the disease.

Course and Prognosis.—In a favourable case recovery, under medical treatment, occurs in the course of from six to twelve weeks. With an operation this time may be rather shorter. Should the child survive, the symptoms disappear completely, although the anatomical changes in the pylorus may remain for some months, possibly permanently.

The death-rate in this disease is very large, varying from 40 to 60 per cent, according to different observers. In these exceptional cases occurring in female children, the symptoms appear less severe and more amenable to treatment than in the ordinary instances in male infants.

In any individual case the most important point in prognosis is the general condition of the child when it comes under treatment. The outlook is better in a patient of 5 lb. in weight than in one of 4 lb. The reason for this is easily understood when the important fact is grasped that the prognosis depends chiefly upon the assimilative power of the intestine. Where emaciation is extreme, the intestinal walls are so atrophic that absorption by them is almost an impossibility. In other words, we can get food through the pylorus by either medical or surgical means, but we cannot ensure its being absorbed. As in simple cases of marasmus, high fever, head-retraction and purpura practically always mean inevitable death.

That the child should at first lose weight under treatment is not a bad sign, but is to be expected. In a favourable case the weight soon becomes stationary, and then slowly increases. A rapid increase in weight in the earliest stages is usually followed by severe diarrhoea, which is very often fatal.

Treatment.—We have first to discuss the indications for surgical treatment in this disease. When it was first described, operative measures were the only ones adopted, and there are still those who advocate their use in every case. It is of course a difficult matter in a disease so dangerous as the one under consideration to decide against a procedure which will ensure the relief of the pyloric occlusion; nevertheless, if a dispassionate view of the whole matter be taken, it would seem that surgical treatment possesses little advantage over medical methods, while it adds a fresh danger, that of shock.

As has been pointed out, in nearly all cases with medical treatment food can be passed through the pylorus in sufficient quantities for the child's needs in the course of four or five days. In other words, what surgery can do in a few minutes, medicine can do in a few days. The difficulty of treatment lies, not in getting food past the pylorus, but in getting it absorbed when it reaches the intestines; and in this, of course, both forms of treatment are equally handicapped. Where the child is so ill as to be at danger of dying within a few days, it hardly seems likely that it can survive the shock of an operation, while the difficulty of getting food absorbed from the intestine and the danger of diarrhoea *ensais*, should the child recover from the effect of the operation. Bearing in mind that the prognosis in this disease depends much more upon the state of the small intestine than upon the condition of the pylorus, it is difficult for one to see how operative means could save life where medical measures would fail.

The indications for operation appear, therefore, to be two-fold. Where at the end of a week or fortnight of medical treatment, properly carried out, the pylorus still refuses to allow food to pass into the intestine in sufficient quantities, as evidenced by the character and bulk of the stools, operation may be deemed necessary. Such cases as these are, however, extremely rare. On the other hand,

where the case is seen very early, before much wasting has occurred, and when therefore the condition of the intestinal walls is presumably good, operative treatment may perhaps be wisely carried out before farther atrophy of the intestine has time to take place.

Of the operations, gastrojejunostomy, pyloroplasty and dilatation of the pylorus, each has its advocates.

Medical Treatment.—The general treatment applicable in all cases of severe wasting must be carried out. Warmth is an essential. Stimulants may be required. Where the condition is very bad, subcutaneous injections of hot saline may be given. Effusions containing 2 per cent of glucose may be tried, but often they are not well retained.

In the treatment of the disease we have to rely mainly upon two measures, namely, gastric lavage and careful dieting; and in making use of these we have ever to be on our guard against—firstly, the accumulation of food in the stomach, from which large and collapsing vomits may occur; and secondly, diarrhoea due to the presence of too much food in the atrophic intestine.

Gastric lavage is the most useful measure we possess in this disease, and is very easily performed (Appendix A). It prevents retention of food in the stomach, it benefits the gastritis which is always present, and by removing particles of food it tends to allay the spasm of the pylorus. A weak alkaline lotion should be used in order to get rid of the mucus in the stomach, and the lavage should be continued until the washings come away clear. Frequently the good effect of lavage is immediately seen in the change of the infant's colour from a dusky grey to a more healthy hue. The lavage must be performed sufficiently often to prevent the accumulation of any large amount of food in the stomach. At first it is best done twice daily, but if necessary it may be done every eight hours for a few days.

The quantity of the food given is of even more importance than its constitution. The utmost care must be taken not to over-feed the child, for over-feeding will set up diarrhoea which is very often fatal. The greatest patience must be exercised, and no attempt made to increase the child's weight. All that should be expected during the early days of the treatment is that the progressive loss of weight ceases. More cases end fatally from diarrhoea than from any other cause. At first, therefore, little more should be given than the smallest amount of food capable of keeping the child alive. To begin with, it may receive one-ounce feeds every hour. The great excess of serum in the gastric juice makes it desirable to give a food that forms a slight coagulum. Such preparations as sterilized milk, desiccated milk, or milk that has been peptonized for thirty minutes, may be recommended. Where possible, breast-milk should be used. It is best drawn off by a breast-pump and administered in a spoon or bottle. Even a non-coagulable food, such as a mixture of whey, cream and raw fruit juice, may, however, become bulky within the stomach when

entangled in the mucus which is secreted in large quantities in this disease.

Should vomiting be troublesome, nasal feeding, as recommended by Dr. Batten, is sometimes of benefit. It probably acts by diminishing the spasm, which may be set up, it is thought, by the act of swallowing. No drugs are of any avail in relieving the spasm of the pylorus.

Any tendency to diarrhea must be carefully treated. Opium is here of considerable value, and may be given with bismuth and soda. When necessary, all milk by the mouth must be stopped, and albumen water or whey given. Rectal lavage may be practised.

These, then, are the principles of treatment in this disease. In conclusion, it may be well to emphasize once more the importance of exercising the utmost patience in dealing with these cases. To attempt to cause a rapid increase of weight in the early days of treatment is but to court disaster.

ACID DYSPEPSIA (*Pyloric Spasm*).

This condition, associated with retention of food in the stomach, vomiting, constipation, and wasting, has been described as due to pyloric spasm. As Dr. Wilcox and I were able to demonstrate, in this class of case there is a greater acidity of the gastric juice than is found in ordinary wasted infants. Inasmuch as spasm plays a part in the production of the symptoms of hypertrophic pyloric stenosis, an entirely different disease, it seems best to term the condition "acid dyspepsia of infants," which has the advantage of suggesting the line of treatment to be adopted.

Symptomatology.—The symptoms are those of vomiting, constipation, and wasting. They usually appear at about the end of the first month of life; but as the loss of weight is comparatively slow, the patient often does not come under observation until it is two or three months old. Either sex is affected. The vomiting is frequent, copious, explosive, and even projectile in character. Constipation is marked. Much pain and screaming, especially after food, are features of this condition. The wasting, as has already been mentioned, is slow at first, and only very rarely simulates that so generally seen in hypertrophic pyloric stenosis. The tongue is as a rule clean. On examination of the abdomen, the dilated stomach is often seen standing out under the abdominal wall, but no peristalsis is visible; nor is there of course a palpable pyloric tumour. The gastric contents show an increase of acid, the total acidity being over 1 per cent, and usually a diminution of ferment activity. There is little or no mucus present in the vomit.

Diagnosis.—From what has been said it will be seen that there are many points of difference between this disease and hypertrophic

pyloric stenosis. A comparison is made between the two conditions in the accompanying table. In difficult cases a few days' further observation may be required in order to exclude with certainty the more serious disease.

	HYPERTROPHIC PYLORIC STENOSIS	ACID DYSPEPSIA (PYLORIC SPASM)
Sex	Usually male	Either sex
Age at Onset	Usually 5 to 14 days	Rather later
Emaciation	Rapid	Slow
Pain	Little	Much
Tongue	Coated	Clear
Vomits	Often projectile	Seldom projectile
Gastric Contents	(Much mucus) Present activity) Total acidity	(No mucus) Present activity) = Total acidity
Gastric Peristalsis	Present	Absent
Typhoid Tumor	Present	Absent
Prognosis	Bad	Good

Table 11.—COMPARISON BETWEEN HYPERTROPHIC PYLORIC STENOSIS AND ACID DYSPEPSIA (PYLORIC SPASM) OR TYPHOID

Prognosis.—With treatment, recovery usually occurs rapidly. In a few cases it is possible that the dilatation of the stomach is so extensive as to cause obstruction from kinking at the pylorus. This, however, is very rare. Death may occur from emaciation, but more frequently from broncho-pneumonia. It has been suggested that these cases of pyloric spasm from acid dyspepsia may become examples of hypertrophic pyloric stenosis. It was with this in view that Dr. Wilkes and I examined the gastric contents in a series of emaciated infants, and for my own part I am quite convinced that the two conditions are entirely distinct.

Treatment.—The stomach, in bad cases, should be washed out once or twice daily. Bicarbonate of soda (2-3 grs.) should be given in the milk-feed, and for medicine some grey powder with soda should be administered. To relieve the pain, a compressive mixture containing sodium bicarbonate, oil volatile, and peppermint water is useful (p. 266).

ACUTE GASTRITIS.

In infants, affections of the stomach are almost invariably associated with changes in the whole of the alimentary tract. In older children acute gastritis becomes more of a clinical entity, and resembles that of adults.

Etiology.—The condition is most commonly due to improper food or feeding, which sets up inflammatory changes. Various general infections, and occasionally drugs, may originate the disease. Its constant occurrence in infantile hypertrophic pyloric stenosis has been mentioned.

Catarrhal, membranous, and ulcerative forms have been described. Most cases conform to the first type. The membranous variety can hardly be distinguished clinically. It may be caused by the Kleb's-Löffler bacillus in cases of diphtheria, but can be due also to streptococci or to the pneumococcus. Ulcerative gastritis is a very rare disease and is usually associated with ulcerative changes elsewhere in the alimentary tract. It has been reported as due to an infection by the *H. pylori*.

Symptomatology.—Vomiting, excessive thirst and some fever are present. Older children may complain of gastric pain and show epigastric tenderness. The tongue is coated and the breath offensive. The vomitus is sour, bile-stained, and may contain streaks of blood. In infants, symptoms from involvement of the rest of the alimentary tract, such as stomatitis, painful dentition, and diarrhoea, are usually present, but in older children these are less constant and less severe.

After one or two days the symptoms tend to lessen, and within a week to pass off altogether. Repeated attacks may produce a condition of chronic gastritis.

Treatment.—Where the temperature is raised, the child should be put to bed. An initial purge is needed. This is often best given, where there is much vomiting, in the form of small doses of calomel, taken hourly until one or two grains have been administered. Gastric lavage is a measure of great value, particularly in the case of young infants. As the tongue begins to clean, a bicarb and soda mixture may be given if the vomiting or pain continues; otherwise small doses of calomel and soda may be employed throughout the illness. For the thirst, sips of tepid water may be allowed.

The diet is of importance. According to the severity of the attack, freely diluted milk may be given, or albumen water or whey may be substituted for milk for a day or two. The return to a fuller diet must be gradual, the condition of the tongue being a guide in this matter.

In infants the stomatitis and diarrhoea will also require treatment.

GASTROMALACIA.

Formerly this condition was regarded as a disease, but it is now known to be merely due to post-mortem changes, depending probably upon self-digestion of the stomach. It is easily recognized if the occurrence is remembered. The wall of the stomach becomes translucent, gelatinous, and so much softened that its rupture is removing the organ from the body is almost unavoidable.

DELAYATION OF THE STOMACH.

Etiology.—In earliest infancy this may result either from hypertrophic pyloric stenosis, or from acid dyspepsia. It may be due to feeding on milk-mixtures which are too large in volume, but I have seen no evidence of its production by feeding on undiluted milk as some opponents of that method have stated is the case. It occurs, together with a paralytic distention of the intestine, towards the termination of cases of marasmus.

Later, it is comparatively frequent during the age of the acute period of rickets, and is particularly common in such rachitic children as suffer from tetany. It is due to chronic gastric indigestion combined with atony of the wall of the organ.

In its acute form it is seen in various infections, particularly in those of the respiratory system. It is found in acute bronchitis, pneumonia, and right heart failure, and is a sign of bad omen in these conditions. Of itself it tends to increase the dyspnea of the patient. Occasionally it causes collapse of some portion of the left lung.

In older children chronic dilatation of the stomach, such as is seen in dyspeptic adults, is not common. It most frequently occurs in neurotic and anæmic children. As Dr. Sutherland has pointed out, it is often associated with cyclical albuminuria.

Symptomatology.—The symptoms are those of chronic gastric indigestion, the only one particularly noteworthy being the occurrence of large vomits.

Diagnosis.—In children, the lowest level of the stomach should be well above the umbilicus. With dilatation of the organ, fullness in the upper part of the abdomen is usually present. Splashing sounds may be elicited. Where difficulty arises in distinguishing dilatation of the stomach from that of the colon, the stomach may be filled with fluid and its area of dullness percussed out. By pouring in a measured quantity of fluid some idea of the capacity of the stomach may be obtained.

Treatment.—In the cases associated with rickets, gastric lavage is the most useful measure at our command. The diet should be

reduced in volume, and small feeds of unflattened citrated milk are of value. The bowels must be kept regularly opened, and as soon as the digestion has improved antacids should be adopted.

Gastric dilatation occurring in cases of pulmonary or cardiac disease may be treated by lavage if the patient's condition is fair. The food should be small in volume and rendered easily digestible by citric acid or pepsinization. Undigested particles of food should be kept from accumulating in the intestine by small doses of calomel. Strychnine is valuable in tending to restore the muscular tone of the alimentary tract.

In older children gastric lavage is rarely necessary. The treatment of gastric dilatation here may be by means of diet, massage, and general measures. The diet should be of small bulk and easily digested. A dry diet, with hot water half an hour before food, may be of value. Massage to the abdomen may be given eight and morning. Various toxic drugs may be ordered, of which strychnine is the most useful, while exercise and fresh air are important means of improving the general muscular tone of the patient.

GASTRIC ULCER.

In children this condition is of great rarity. Ulcer of the stomach may be found in the new-born, where it is usually associated with a general septic infection. Ulcerative gastritis has already been mentioned (p. 273). Tuberculous ulcers are so rare as to be only of pathological interest.

HÆMATEMESIS.

This is an uncommon condition in children, even taking all cases, both true and spurious. In acute gastritis, or the coming of severe cases of marasmus, small flecks of dark blood are sometimes seen. In the new-born, and in septicæmic conditions, hæmatemesis is occasionally seen. It may occur in hæmophilia, scurvy, purpura, acute leukaemia and in primary splenomegaly.

Spurious hæmatemesis is of relatively common occurrence, and its possibility must always be borne in mind. In babies at the breast the blood may come from a fissured nipple. In other children blood may be swallowed as the result of bleeding from the nose, gums, or pharynx. In those rare cases of profuse hæmoptysis from tuberculous or gangrene of the lungs, the stomach is usually found post mortem to be filled with dark clotted blood.

V—DISEASES OF THE INTESTINES.

CONSTIPATION.

Etiology.—Constipation may be a prominent symptom in several diseases which require mention. In infancy it may be due to a

malformed anus or rectum, to congenital dilatation of the colon, or where associated with persistent vomiting, to pyloric spasm from acid dyspepsia, or to infantile hypertrophic pyloric stenosis. At an older age it may be caused by painful conditions of the anus, such as fissure or hemorrhoids, or, especially if alternating with diarrhea, by rickets or abdominal tuberculosis. Another cause at any age is intracranial disease. It should be remembered also that constipation may be a very troublesome symptom in cases of mental deficiency.

We are less concerned in particular with constipation as it occurs apart from any other disease. In such a case many factors may be responsible for it. It is a condition to which breast-fed infants are particularly prone, and in them is probably usually due to a lack of fat, with possibly an excess of protein, in the milk. In bottle-fed children the same deficiency is responsible for the same symptom, while irregularity and other faults of feeding may also be present. In older children, a dietary containing bulky and indigestible articles of food, often given with the idea of promoting regular evacuation of the bowels, is a common source of constipation. A deficiency of fluid is a frequent dietary error. Undue dryness of the intestinal contents is both a cause and effect of constipation. A diminished secretion of the intestinal glands predisposes to this.

Loss of tonicity of the intestinal muscle is again both a cause and a result of constipation, and in cases of long standing is usually the most important factor in causing the continuance of the symptom. With it there may be enlargement of the abdominal muscles due to malnutrition or rickets.

Lack of exercise and fresh air are predisposing causes in some cases, while lack of training, whereby the habit of a regular evacuation of the bowels is inculcated, may be responsible for constipation in older children.

Spastic Constipation.—In a few cases the colon, instead of being atonic, is rigidly contracted, and can be felt as a thin tight rod crossing the loins of the pelvis. Such a condition should be excluded before treatment on ordinary lines is adopted, for this form of constipation requires different measures to relieve it. For this reason, therefore, spastic constipation is described separately (p. 280).

Symptomatology.—The normal character and frequency of the stools during infancy are described on p. 21.

Where constipation arises acutely, and especially where accompanied by gastric disorder due to some improper food, symptoms may result closely resembling those seen early in enteric fever. The temperature is raised, even up to 103° or 104° , the tongue is furred and the breath foul. The patient is apathetic, although irritable when disturbed. As the result of a purge these symptoms quickly disappear, and often the diagnosis from typhoid fever can only be made by noting the result of this treatment. The motions passed, scybala at first, become soft, highly offensive, and contain undigested food.

In chronic cases, where the bowels are opened perhaps every two or three days, or possibly inadequately opened daily, the child may show very little constitutional change. It is remarkable how well a child can keep, even when the bowels act only once in five or six days. In infants constipation may be masked before it interferes with a normal gain in weight, but it is, however, a frequent cause of failure to gain weight, or of actual loss of weight. In older children constipation may be recognizable at sight: the sallow complexion, sunken dark-ringed eyes, coated tongue, and foul breath being very characteristic. Thread-worms are often complained of by constipated subjects. The abdomen is rather protuberant, particularly along the course of the colon, which is usually dilated. Constipated children, although able to run about well, are prone to be languid and easily exhausted. They may suffer from disturbed sleep, in atonic subjects night-terrors may be present. The appetite is capricious rather than uniformly poor. The temperature at night may be raised about a degree, evidence of the toxic absorption which results from exercise.

Treatment.—From what has been said on the etiology of constipation, it will be seen that there are several matters which require consideration for the successful treatment of the condition. We have to endeavour not only to relieve constipation, but to cure it,—often a very different matter, requiring prolonged and careful treatment.

General Treatment.—Mention may here be made of the good effects of training, fresh air, and exercise.

Massage along the course of the colon is a very valuable measure, and should be ordered in all cases of severity in which the intestine is atonic. By this means peristalsis is assisted and the loss of tonicity of the muscles of the intestine and abdominal wall is corrected. In slighter cases abdominal massage alone is often productive of a permanent cure. It should be carried out night and morning, the massage being given along the course of the colon from caecum to sigmoid. If done by an inexperienced person, it is advisable that a little oil should be used as a lubricant.

Dietetic Treatment.—Mild cases of constipation in infants may be benefited by the addition of barley-water, brown sugar, or cream to the diet.

In order to increase the fluidity of the intestinal contents, plenty of water should be given in the diet, and the child encouraged to drink freely both at and between meals. The same result may be obtained by the use of the saline aperients.

In very mild cases in older children constipation may be benefited by the addition to the diet of substances which leave a large residue within the bowel and so stimulate peristalsis. Of these the most useful are porridge, whole-meal bread, and raw apples. A word of warning must however be given in connection with this method of treating constipation. It is only applicable to mild cases, for in those

of any severity the intestinal muscle is probably atonic: it cannot, that is to say, deal with the contents of the bowel as they are, and the addition of further material to them will only make the constipation worse. In children the dietary is relatively much richer in vegetables than it is in the case of adults, so that such measures as these are not usually required. It is very common to hear of apples, bananas, and currants being given for constipation. These may of course set up an acute indigestion and so cause the bowels to act, often much too freely, but it is more usual to find that the constipation grows gradually worse under their use, and, as has been explained, such a result is only to be expected in cases of any duration or severity. Doubtless it is frequently true that "an apple a day keeps the doctor away," but when it does so it is only too often at the expense of the patient's health. Of measures for increasing the bulk of the intestinal contents, the most successful is oatmeal in the form of a thin porridge.

Such fruits as stewed prunes and figs, or their juices, come under rather a different category, as they act by some contained aperient principle. Rose-leaf juice has a mild laxative effect.

Drug Treatment.—We are here handicapped by the fact that many aperient drugs are very nauseous, and, unfortunately, some of the most efficacious are the least pleasant in taste. It is necessary therefore to consider the taste of these drugs in mentioning their respective values. It would be a useful lesson to home-physicians I sometimes think, if they were called upon to administer the drugs they so light-heartedly prescribe for their children patients.

As occasional purges, or for acute conditions, calomel and castor oil are the best. The former may be given as a powder with a little white sugar, and is almost always well taken. In a few children, however, all powders seem to set up vomiting. For a baby of a year old a grain may be given in one dose, or in fractions quickly repeated. If cold, he present a grain or two of sodium bicarbonate may be added. Castor oil is not so well taken, for many children object to it strongly (as well they may), and will always succeed in vomiting it if they be forced to swallow it. Few of the "flavourless" brands are very satisfactory. It is best given in a little milk, flavoured perhaps with lemon-juice and sugar. In this form it is taken by most children without great difficulty. A drachm may be given to a baby of one year. It generally acts very well. It is of particular value where the constipation has been preceded by vomiting or is associated with toxic symptoms. It is not to be recommended for cases of simple chronic constipation owing to its later sedative effect upon the intestine.

The compound powder of liquorice is very certain in its action as an occasional aperient, but gives rise to some gaging. Its chief drawbacks are its bulk and appearance, on account of which some children cannot be made to take it under any pretext.

The collections of soda and sulphur are, as a rule, well taken, and act well. Very usually a mixture of equal parts of the two may be

ordered, half a drachm being given to a child of three or four years old. Syrup of senna is pleasant to take, but is a little uncertain in its action. It may be given in doses of a drachm for a baby one year old, and half a drachm for a smaller infant.

Rhubarb may be of service when a secondary astringent effect is wanted, but it is not easily taken by children on account of its taste. For a child of one year, 7 grains of powdered rhubarb may be given with some soda salt tingedness in a mixture, or as a powder with calomel and soda.

The drugs which may be given for a prolonged period for the relief and cure of chronic constipation, such as is associated with atony of the intestinal muscles, must now be discussed. Where regularly given, medicine is generally well taken by children, who cease to object very strongly after the first few days. Many will take out of a medicine-glass what they will refuse from an ordinary cup.

In chronic cases it is well to start with a purgative such as calomel, or with a rectal injection of glycerin. In very obstinate cases enemata or suppositories of glycerin or of soap may be used at first; but owing to their irritating effect upon the mucosa of the rectum, they should be discontinued as soon as the bowels act regularly without them.

In babies in the first few months of life various drugs may be of use. In very mild cases magnesia, or a drachm of magma dissolved in milk, may be given. Slightly more powerful is the popular remedy of olive oil, in doses of from half to one drachm. A useful remedy in mild cases is sodium phosphate, five to ten grains of which may be added to the baby's bottle once or twice a day. Grey powder may be given for many weeks; it is of great value in slight cases and as an adjunct to other measures in cases of greater severity. A little sodium bicarbonate may be added where colic is present.

A particularly valuable remedy is the infusion of senna pods. It is almost tasteless, and nearly always well taken; it is readily prepared, and its doses are easily graduated. It acts without causing griping, and is fairly certain in its results. Where used continuously it often effects a complete cure. The method of preparation is given in Appendix A. It should be given freshly prepared. For a child of one year the infusion from three pods (making a two-drachm dose) may be given at first, followed by half doses nightly. For a younger infant smaller doses may be given. For a child of eight years old an infusion of four or five pods will probably be necessary.

In stubborn cases a mixture may be of more value, but it is less pleasant to the taste. It should usually contain some saline aperient in order to attract fluid into the bowel combined with drugs which improve the tonicity of the intestine. Such a prescription as the following may be given to a child 2 years old:—

R. Ext. Casc. Sag. Liq.	℞ss	Tinct. Belladonna	℞j
Sol. Sulphat.	gr. x	Syr. Zingib.	℞xx
Tinct. Nuc. Vom.	℞ss	Aq. Ment. Pip.	ad ʒij.

This may be given once, twice, or three a day, as necessary. In order to render it more efficacious, 10 to 20 minims of the syrup of senna may be added. Still more successful are the tinctures of aloes and podophyllin. The former is, however, extremely unpleasant, and both are liable to produce a good deal of pain. For a child of one year four minims and two minims respectively may be given. In very wasted infants the saline aperients are of less value than in healthier patients, as here they obviously act under a disadvantage.

In older children some such mixture as has been just mentioned may well be given, the doses of the ingredients being increased. Cassia with malt is a very useful preparation. The saline aperients given alone are of use in the slighter cases, but are not to be preferred in the more stubborn instances to mixtures containing in addition drugs acting on the bowel-wall. The sulphates of magnesium and sodium may be given with some syrup and peppermint-water. Aperient water is useful; half a wineglassful may be taken before breakfast. The infusion of senna pods, as already mentioned, may be given to older children as well as to infants.

In such treatment as has been suggested, it is of great importance that the medicine should be taken for a long time. The doses may be gradually diminished, but small doses should be taken for several months. The drug treatment should be combined with other measures already mentioned, namely, exercise, fresh air, abdominal massage, increase in the amount of water taken, and possibly some change in the diet.

SPASTIC CONSTIPATION.

Spastic constipation with spasm of the colon is far less common than the atonic type. In the *fecal fossæ* the faeces can be felt, and spasmodic contraction can readily be appreciated. This form of constipation is met with usually in neurotic subjects, and also in some cases of intracranial disease.

Treatment.—The measures relieving the atonic cases are corroborated here. Abdominal massage, drugs stimulating the intestinal muscle, and food leaving a large residue will all do harm. The treatment which is most successful is that which is directed towards keeping the intestinal contents small in bulk and soft in consistency, and towards relieving the spasm. Thus the saline aperients are very useful combined with belladonna in large doses.

"MUCOUS DISEASE."

Under this name, Dr. Eustace Smith has expressed a form of intestinal indigestion, usually with constipation, in which mucus is passed in the stools. As it forms a convenient label for an important group of cases, we may adhere to the title by which the condition is now well known.

Symptomatology.—The characteristic of "mucous disease" is the type of motion passed by the patient. In it there is much mucus. Scybala are often present coated over and surrounded by mucus, but in other cases the motion is soft and uniform, and contains masses of mucus.

The symptoms, it may be pointed out at once—for this is the value of emphasising the condition—are such as may easily give rise to a suspicion of early tuberculosis. The child becomes pale and loses flesh, the eyes show dark rings round them, the appetite is poor or capricious, and the child seems definitely "out of sorts," without there being anything to account for it. The patient is usually of a neurotic type, and quickly becomes tired with playing. The temperature at night is slightly raised. The tongue is faddy, coated, and often shiny. There is a good deal of abdominal discomfort, with slight colicky pains. Restlessness at night is frequent; sleep may be disturbed by frightening dreams. Recurrent "bilious attacks" may occur.

Diagnosis.—Such an indefinite malady as this may well be mistaken for the beginnings of abdominal tuberculosis; or should a cough from tonsillitis or bronchitis be present, an early pulmonary infection is feared. No definite physical signs in the abdomen or lungs can be elicited, but the child is given much cod-liver oil and gets, as a result of this, gradually worse.

The diagnosis is easily made, if the condition is known, by the examination of the stools and the result of correct treatment.

Treatment.—The amount of carbohydrate in the diet should be limited. All sweets should be stopped, and only as much sugar should be allowed as may be required to render the food palatable. For drugs, aperients should be given, even where the motions are uncoloured. Of most use are such preparations as those of rhubarb and soda in a mixture or as a powder. Nux vomica may often be advantageously combined with them. Calomel with soda should usually be given at first for a few nights with the other drugs. As the stools become normal, and the colour and appetite return, tonics may be given. Later cod-liver oil may be beneficial.

One of the most important points in connection with treatment of "mucous disease" is the recognition of the fact that cod-liver oil and tonics are useless and often harmful, until the intestines have been put in order by a course of aperient drugs.

DILATATION OF THE COLON.

This is now an uncommon condition in children. In its simplest form it is seen in association with constipation, atony of the intestine being both a cause and effect of confinement of the bowels. To this type we need not allude here, for the subject of constipation to which

it properly belongs has been already fully discussed. There are in children two other conditions which must be described: firstly, Hirschsprung's disease, in which the large intestine is greatly dilated; and secondly, meconium colic, in which the dilatation of the colon is of far less degree, and is probably associated with some dilatation of the small intestine.

HIRSCHSPRUNG'S DISEASE.

(Congenital or Idiopathic Dilatation of the Colon).

The condition known by these terms cannot as yet be looked upon as very clearly defined, and for this reason it seems better to allude to it as Hirschsprung's disease, rather than to tie ourselves down to the limiting options of congenital or idiopathic.

It is a rare condition of which the cause is not yet known. In some cases there seems to be stricture or spasm of the anus with hypertrophy of the sphincter; in others there are intestinal adhesions, but in the majority no such possible causes are recognizable. In all, however, there is severe constipation, but to what extent this is a cause or an effect of the dilatation of the colon cannot as yet be settled.

Again, while it is difficult to be sure that any of the cases are truly congenital, it is hardly possible to regard all of them as being certainly due to an anatomical malformation present at birth. As will be mentioned in dealing with the symptoms of the disease, there is a group in which the symptoms arise at or very shortly after birth, and such may possibly be actually congenital. Even here, however, we have to remember that, apart from abnormal conditions of the anus, the colon in earliest infancy is long and tortuous, and that this may be the origin of constipation to which the changes in the colon are secondary. But judging from the seriousness of the condition seen in infancy, it is difficult to imagine that the instances of severe dilatation of the colon seen in older children are in quite the same category.

While we have to describe here various classes of cases which have much in common with one another, it seems preferable to do so under the heading of Hirschsprung's disease, and to make no reference to the nomenclature of the condition to any theory of its causation, which is at the best doubtful.

Symptomatology.—The prominent symptoms are constipation and abdominal distention, to which must often be added severe wasting.

(1) In small babies, these symptoms arise directly after birth. The constipation may be extremely severe, so that the bowels remain unopened for periods of one or two weeks. The distention may be marked; it diminishes considerably if the bowels are freely opened. There is very great emaciation. An important symptom in this group is the severe and often fatal collapse which may ensue after the passage of a large motion, analogous to that which follows a large vomit in cases of infantile hypertrophic pyloric stenosis. In many cases

peristaltic waves along the course of the colon may be clearly seen through the abdominal wall. The sigmoid may stretch over into the right flank before entering the pelvis, so that left-to-right peristalsis may be visible. In a few cases obstruction and peritonitis from stercoral ulceration of the bowel cause death, but more often this is due to emaciation, with possibly a terminal syncope following the passage of a large motion. This infantile type of case is nearly always, if not invariably, fatal.

(2) In another group the patient comes under observation for the same symptoms at a rather later age, from two to five years old. Here there is constipation alternating with the passage of very large and loose motions. The abdominal distention and peristalsis are usually more marked than in the former group. After an action of



Fig. 14.—DILATATION OF THE COLON.

the bowels the girth of the abdomen is much reduced for the time. As Dr. Langmead has pointed out, these children very commonly show tetany, facial irritability, and, rarely, laryngismus stridulus, such attacks lasting over a long period and showing a great tendency to relapse. Here too a fatal ending is the rule, although it may be postponed for many months.

(3) In still older children, and even in young adults, a similar condition of the large bowel is met with, although there seems little tendency to tetany and the other nervous signs mentioned. Here the prognosis seems distinctly better. Although there are recurrent attacks of constipation, distention, and illness the patients appear to survive.

The relationship of these three groups of cases to each other I do not attempt to explain, merely setting them down as I have seen them.

Morbid Anatomy.—As a rule the whole of the large bowel is affected (Fig. 35), but the changes may be limited to the sigmoid. In the affected part the bowel is enormously dilated, its walls are thickened, and its mucosa reddened and thickened. In fatal cases large masses of fecal material are found within the bowel, often adherent to, and occasionally producing ulceration of, its wall. In *Solters* these masses are hard in consistency and bright yellow in color. The other changes already noted may be present.

Diagnosis.—This is a matter of difficulty in very young subjects. Where suspected, the capacity of the colon may be gauged by the injection of warm saline solution. Where peristalsis is present, the condition may be mistaken for hypertrophic pyloric stenosis, as in



Fig. 35.—Hirschsprung's Disease.

both diseases left-to-right peristalsis is seen. In Hirschsprung's disease the waves pass at a low level across the abdomen, while the symptoms are fecal rather than gastric. In older subjects the very large stools and the marked decrease of the abdominal distention following an action of the bowels are very characteristic.

Prognosis.—Sufficient has been said on this subject under the symptomatology of the various groups of cases described.

Treatment.—In young infants this is a matter of great difficulty. There is serious constipation, and when the bowels are opened severe or fatal collapse may ensue. For this reason, therefore, operations must be used cautiously, and be confined with rectal irrigation rather than

enemas) and abdominal massage. In older children the same lines of treatment are to be used, but with greater freedom than in infants. The febrile and other nervous symptoms subside with the use of rectal irrigation and sedatives.

Operative treatment has been advocated, but its success in the various forms of the disease in infants and young children has not as yet been great.

MORBUS CÆLIACUS

(Cælia).

This condition may be conveniently considered here, as the very large stools characteristic of it suggest at once some dilatation of the lower bowel. That there is any definite pathological entity known as cæliac disease is very doubtful, yet the condition is of clinical interest owing chiefly to the ease with which it is mistaken for abdominal tuberculosis. It is probably associated with a general dilatation of the intestines, both large and small being involved. I have never seen an autopsy made upon such a case.

Symptomatology.—The condition is seen most frequently in children of four or five years of age, but it is not of common occurrence.

The chief characteristic is the condition of the stools. They are large, frothy, pale, unformed, and greasy; of the colour and consistency of porridge. The bowels act once or twice in twenty-four hours. The condition is really one of diarrhoea,—that is, excessive intestinal elimination,—without frequency of defecation.

The abdomen is large and shows general distention. On palpation it is extremely soft and doughy, and no enlarged glands nor signs of acutis are present. The constitutional symptoms are slight, namely, pallor, coated tongue, and only a very moderate amount of general wasting. The appetite is, as a rule, fair or good.

The course of the disease is prolonged. It is difficult to cure and relapses are common. It is not dangerous to life.

Diagnosis.—The general abdominal distention and the slow improvement under treatment render the condition liable to be easily mistaken for abdominal tuberculosis. The very soft feel of the abdomen, the characters of the stools, and the absence of definite signs of tuberculosis are, as a rule, sufficient to prevent such an error being made.

Treatment.—At first the child should be kept in bed, and high irrigation of the colon with warm saline practised once or twice daily. In this condition the latter is the most valuable means of treatment that we possess. After an initial purge, bismuth salicylate or salol

may be given, but drugs are here of minor importance. The diet must be plain, light, and easily digested. As the motions become firm, abdominal passage may be ordered.

COLIC.

The most troublesome form of colic in children is that which occurs in constipated infants, and is often associated with acid dyspepsia (p. 271). In these cases, screaming occurs for hours day after day, or—when it is still more noticeable—night after night. It may be that the food which is being given is insupportable in quantity or quality, or that it is administered at irregular intervals, or too quickly. It may also be due to the disengagement following upon constipation. The mother, however, is likely to infer that the irrepressible noise is due to hunger, and the child is thereupon fed again. Though lalled momentarily by this, its screaming is soon renewed with increased energy.

In older children colic is most commonly due to some error of diet, and frequently precedes or accompanies an attack of diarrhoea.

Diagnosis.—In babies the pain of colic is accompanied by the drawing up of the legs, so that the knees are flexed upon the abdomen. The characteristic sign that pain is due to colic is the relief afforded by the passage of feces or flatus.

There are many other causes of screaming in babies than colic. Hunger, thirst, cold feet, and wet napkins are the most common. Painous or hyperacidic of the uræ may cause screaming associated with incontinence. During the second half of the first year of life curvy may be the cause. Occasionally in meningitis there may be sudden and short attacks of screaming. Intussusception is another serious cause of screaming. In mentally deficient children screaming may be almost constant, and the child may be brought to the doctor on this account.

Treatment.—Any errors in the feeding of the child must be corrected. Regular feeds of a suitable nature and properly given must be insisted upon. Often the cause of the milk is at fault, but by the use of aperients and alkalis the tendency to rick can be remedied without the strength of the food having to be reduced. The milk may be given with the addition of some sodium bicarbonate, or may be rendered more easily digestible by sodium citrate.

For drugs, aperients such as glycerine, or small doses of calomel with soda, are of great use. Such should be given regularly two or three times a day. For the attacks of pain, poultices or a tight binder may be applied to the abdomen. The child should be kept very warm. A carminative mixture may be given, such as—

R. Sod. Bicarb.	gr. ij.	Sp. Chloroform.	℥j.
Sp. Annon. Ararat.	℥ss.	Ag. 5204.	ad 5j.

A couple of drops of laudanum in warm water is an almost certain remedy, but is obviously not such as should be given where it can be avoided. In most cases, perpetually recurring colic ceases under treatment by aperients and alkalies.

In older children colic is usually best treated by a dose of castor-oil.

ACUTE DIARRHŒA.

Any attempt at the classification of acute diarrhœa in infants is rendered of small value by the fact that there are few pathological changes characteristic of any clinical types that may be described. Nor would it be of any advantage to separate widely different classes of diarrhœic conditions, for in infancy the disease is so liable to change rapidly from one type to another. An exception must be made of dysentery, a condition of definite symptomatology and morbid anatomy. To some extent, also, the epidemic type, the "cholera infantum," or epidemic enteritis, can be separated from other cases on clinical grounds. Where a strong and healthy infant is suddenly seized by the disease, and is killed by it within a day, we seem to have a very distinct class of case; but the separation of such is rendered less successful by the fact that many a mild attack of diarrhœa may terminate in a condition exactly resembling that of an epidemic case, and the same may also be said of the diarrhœa which may cause the death of an infant who has been suffering for months from chronic diarrhœa associated with marasmus.

We shall here, therefore, in dealing with acute diarrhœa in infancy, divide the cases into mild, severe (including epidemic cases), and, thirdly, those of dysentery.

1.—MILD DIARRHŒA.

Slight attacks of diarrhœa in infants are due, most commonly, to errors of diet, often of a trivial nature, but there are other causes operating directly or indirectly. Rickets, syphilis, and wasting, are important predisposing factors. The influence of rickets in confining to attacks of diarrhœa, as of bronchitis, has been emphasized in the account of that disease. Again, chills or slight attacks of fever may bring on looseness of the bowels. Such may also be attributed to sudden changes in the temperature, or to the barometric pressure of the air.

In older children over-feeding and the eating of indigestible food are the most common causes of diarrhœa. Some of the specific fevers, notably scarlatina and measles, may be accompanied at the onset by an attack of diarrhœa, often more than mild in nature.

With only a slight looseness of the bowels, the symptoms may be practically none. The stools are more frequent than normal, offensive, and become liquid or even watery. In infancy they usually contain curds, and often small particles of mucus. After the first day or so

they are frequently green in colour. The child becomes pale and irritable, the tongue is coated, and the breath foul, and often there is vomiting in addition. There are generally slight fever and much thirst, and the child begins to show some hollowing round the eyes and "pinching" of the nose. As the attack subsides, the stools become less frequent and less offensive, the appetite begins to reappear, and the child quickly picks up strength again. There is no little tendency for any sudden or unaccustomed exercise or any error in diet to cause a recurrence of the trouble. On the other hand, either the attack or the relapse may develop into a much more serious form of diarrhoea.

The pathological changes in mild cases, and indeed in all cases of acute diarrhoea, with the exception of dyscolitis, are insignificant. There may be some injection of the mucosa of the alimentary canal, with swelling of Peyer's patches and the follicles of the colon. There is some excess of glandular secretion on the living surface of the intestine. The mesenteric glands are usually large, white, and rather soft.

Treatment.—The treatment to be adopted in such cases as are here being considered is simple. Rest in bed is one of the most useful measures to adopt, as it tends to lessen the frequency with which the bowels act, and also to diminish the amount of toxic substances absorbed from the disordered intestinal tract. An aperient should be given at the onset of the attack, preferably such as will exert a secondary astringent effect, such as castor-oil. A full dose of the oil may be given, followed by the regular administration of a castor-oil mixture:—

R., Oil Ricini	Rv	Tragacanth.	gr. i
Glycerini	Rv	Aq. Ment. Pp.	℥i 5j

In children past infancy a mixture of rhubarb and soda is useful:—

R., Pulv. Rhai	gr. ʒi	Syrup. Zingib.	℥vii
Sod. Bicarb.	ʒss	Aq. Ment. Pp.	℥i 5j

If the diarrhoea continues after the tongue has become clean, bismuth with or without opium should be given:—

R., Bismuth. Carb.	gr. x	Tragacanth.	gr. i
Sod. Bicarb.	ʒr	Sp. Chloroform.	℥ii
		Aq. Cam.	℥i 5j

The milk may be rendered more easily digestible by dilution, or by peptonization. When the coating of the tongue has disappeared and the appetite has improved, ordinary diet is gradually resumed.

Should the attack be long in clearing up, or should it threaten to become more serious, some of the measures recommended in the treatment of severe diarrhoea should be ordered, particularly that of renal irrigation.

7.—SEVERE DIARRHOEA.

(Including Epidemic Diarrhoea.)

This may develop from a mild attack, or may terminate the chronic diarrhoea of a malarious infant—but, and these are the cases that most deserve the term "epidemic," it may attack a strong, fat child. In whichever of these ways it begins, the resulting state is the same, and so here only one description is given—that of the condition frequently termed "cholera infantum."

Etiology.—Of its etiological factors some are known, while others are as yet unsettled. It is commonest in the hot weather, particularly during a spell of intense heat which has been preceded by warm weather. The incidence of the disease is much higher in bottle-fed infants than in those fed at the breast. Again, it is abundantly clear that this is a disease occurring among the poorer classes in large towns—that is to say, it is associated with ignorance, over-crowding, dirt, and bad sanitation. It is caused, in short, by dirty milk. While the vast majority of patients are under one year of age, older children and even adults may suffer. There can be no doubt that the disease is of bacterial origin; but what micro-organism is concerned is still unsettled. There seem no grounds for supposing that the disease in England is due to a dysentery-like organism, as is said to be the case in America, nor are the conditions in the two countries clinically the same, the cases here simulating cholera, while in America they resemble dysentery and seem to be more analogous to the class which is described later as *discolitis* (p. 204). Dr. Morgan has made a very lengthy investigation into the bacteriology of the disease, and the bacilli which are associated with his name may prove to be the causative agents.

It must be emphasized, however, that diarrhoea of the epidemic type is not a local but a generalized systemic infection. It may be that the septicæmia is due to some intestinal organism which is the cause of the diarrhoea, or that in the disordered state of the bowel some other organism gets into the circulation from the alimentary tract: but, on the other hand, it is quite possible that the blood infection is the primary condition, and that the intestinal symptoms are the result, and in no way the cause, of the septicæmia. It must be admitted that the problem of the actual cause of the disease is, as yet, far from being solved.

Symptomatology.—The symptoms will be described as they occur in a previously healthy infant. Where the attack has been preceded by chronic diarrhoea, the wasted condition of the child modifies to a slight extent the clinical picture, but it is usually easy, by looking at the patient's face, to recognize the occurrence of an acute attack succeeding chronic diarrhoea.

The number of the motions passed is much increased and may be as large as twenty in the twenty-four hours: from eight to twelve

is quite common. At the onset of the attack the first two or three stools are usually yellow in colour, offensive, soft, and containing curd. In a few hours they become green and watery, losing their offensiveness gradually and acquiring a sour odour. Curds are still present. Later, as the disease progresses, the motions consist of a watery evacuation with small pieces of green matter in them. From this they often pass to the state described as the "rice-water stool," a watery, pale, almost colourless evacuation, containing only white shreds of solid material. Should the child live, and the condition improve, the stools become less frequent, and are usually green and rather watery, but after a few days they lose their green colour and become a pale yellow—this being a sign of definite progress. Blood is rarely seen in the motions of these cases. Vomiting is nearly always present, but is often fairly easily controlled by treatment. The vomitus contains curds and mucus, and, in a few cases, small flecks of dark blood. This last means a very severe attack, and usually forbodes a fatal end. From the acid nature of the stools there is usually much soreness of the buttocks, the epidermis of which may be shed.

The further symptoms are referable to the loss of fluid occasioned by the diarrhoea, the collapse induced, and the septicæmia present. Thus, the eyes are sunken, the anterior fontanelle depressed, and there is great thirst. The skin is described as mottled, by which it is not meant that the skin is not easily extensible, for it is more so than in health, but that when it is pinched up in the fingers it returns into position slowly. This change in the skin, and the loss of fluid from the tissues round the eyes, differentiate a condition of acute diarrhoea from one of simple chronic wasting. The urine is diminished in amount, and shows, as a rule, a distinct cloud of albumen on boiling, and frequently gives the reactions for acetone. The collapse is profound. The infant is cold; the face pale, often grey; the temperature may be considerably subnormal; the pulse is feeble and fluttering, or imperceptible; the respirations are shallow. One of the best tests of the infant's condition is the character of the cry; in bad cases it is merely a feeble whim. The eyes again are a guide; in the most severe cases they are covered with a film, and if they are bright, and especially if the infant looks at objects around it, there is some hope that recovery may take place. The symptoms of the septicæmia may be those of head-contraction, general rigidity, and convulsions. The temperature, if the collapse be not too great, may be up to 104° , or even higher. Sooner or later oedema appears on the dorsal surfaces of the hands and feet, or it may be seen all over the body and round the eyes, giving a facies resembling that of acute nephritis. Occasionally this may be seen early in the disease; but there is even here, as a rule, no inflammation of the kidneys. Sometimes urticaria is seen. Purpura, nearly always starting as small petechial hæmorrhages on the lower part of the abdominal wall, appears towards the end of severe cases, and is almost invariably of fatal significance.

The disease, as it is seen affecting previously healthy infants, is one of most acute onset. Suddenly the child vomits, and may be convulsed, the bowels are frequently opened, in a few hours watery stools are passed, and the infant lies at the point of death, collapsed and poisoned. Where a wasted infant is attacked, the convulsions and rigidity develop, the diarrhœa becomes of the choleric type, the eyes sink in farther than before, and death often takes place with hyperpyrexia.

Morbid Anatomy.—There are no changes in the alimentary tract by which the condition can be recognized, and the separation of the case is made on clinical grounds. It is not impossible, however, that the determining factor may in the future prove to be, not that the infection is due to some specific organism, but that in this group of diarrhœic cases there is an infection of the blood-stream. The mucous membrane of the intestinal tract shows areas of infection and inflammation, usually of a very insignificant character. Occasionally there is some hæmorrhage into the submucous coat of the stomach, and exceptionally there are small follicular ulcers in the lower end of the ileum. The Peyer's patches are nearly always found swollen and a little injected, as is so commonly the case in autopsies on children. The follicles of the colon are not, as a rule, much swollen, unless the acute attack has been preceded by chronic diarrhœa. The intestines may be empty, or may contain a small amount of green slime. The chambers of the heart contain but a very little blood, which shows post-mortem clotting. The liver and spleen show no obvious changes unless there has been antecedent chronic diarrhœa, in which case the former is pale and often fatty. The mesenteric glands are swollen, soft, and pale. The brain is anæmic, and usually the meninges are serfamous. The middle ear contains some mucus, as is the case in practically all children under eighteen months of age whatever be the cause of death, but the membranes are seldom inflamed (p. 112).

Diagnosis.—In a well-marked case the diagnosis can be made at a glance. Occasionally the nervous symptoms of head-retraction and rigidity suggest a case of post-hæic meningitis starting with diarrhœa, but the condition of the fontanelle (collapsed instead of taut) is, as a rule, sufficient to prevent mistake. The occurrence of focal oedema simulating that of acute nephritis has been mentioned, and only needs to be remembered to stop a diagnosis being made without a close investigation of the urine.

Prognosis.—The epidemics of diarrhœa vary in their severity in different years, for the most part in accordance with the atmospheric temperature. In a bad year only a very few of the really severe cases recover. The author has had abundant opportunity for watching a large number of cases treated with every resource and nursed with

antising acid, but yet, taking all cases together, yielding a death-rate of nearer ninety than eighty per cent. In wasted infants, diarrhoea of the epidemic, or as it may be called, the septicaemic type, is almost invariably fatal. Even in strong children the disease is one of the greatest danger, the results of treatment are exceedingly bad, and the great majority of those attacked die. At the beginning or end of the diarrhoea season the prognosis is better and the chance of recovery is considerably improved. In a disease of such fatality it is of small service to point out symptoms of special danger. Purpura is a sign which practically invariably means a fatal ending; coelestia is slightly less serious. Earlier in the disease the colour of the child and the reaction to treatment during the first few hours are of some use in forming an opinion as to the ultimate outlook of the case; but it is of great importance to remember that with an initial improvement, a relapse proving fatal often occurs towards the end of the first week; more cases die at that time than during the first twenty-four hours of the disease.

Treatment.—Prophylactic Treatment.—The importance of this can hardly be exaggerated, for at the present time we must acknowledge that epidemic diarrhoea of the severe type which is common in London is a disease which medical skill is unable to combat, and which carries off its thousands of victims each year. When we consider that during the three summer months of a hot year (1906, for example) 14,000 infants under one year of age die of diarrhoea in the large towns of Great Britain, we must surely grasp the fact that it constitutes a great danger to the country, particularly so now that our birth-rate is falling. As has been emphasized already, it is not an epidemic which carries off only the wasted and diseased infants, but it is one that attacks and kills in large numbers the well-nourished and apparently healthy children. The attitude affected by so many that the disease merely eliminates the unfit has not even the support of scientific accuracy.

We have, then, to consider the problem of feeding the children of the poor with a sterile milk, and by this is meant that the milk actually given to the baby is sterile. With the poverty, ignorance, and dirt found in the homes of the poor, it is obvious that sterility of the milk can only be assured in one way, that of boiling every feed in the bottle from which the infant is going to be fed. This plan also has the advantage of doing away with that bogey of all sterile feeding, infantile scurvy; for, as has been stated when dealing with that disease, the alkaline salts rendered insoluble during the boiling are not lost to the child in this method (p. 74). The late Professor Babin carried out this plan with the greatest success in feeding a large number of infants. In his "consultations" he adopted the method of distributing to mothers milk which had been boiled for forty-five minutes, it having been sterilized in bottles containing enough for one feed only; thus ensuring, as far as possible, that the baby was actually fed with milk that was sterile. To those who could pay, these bottles of milk were

sold at a small profit, while to others they were given at a reduced rate, or even free. But such a consultation, carried on with strict medical supervision, was self-supporting. A full account of the work is given in Bodin's "The Nursing," which has been translated into English by Dr. W. J. Maloney. This scheme has been adopted with some modifications in certain of the boroughs of London and at some provincial towns, and with great success; but still much prejudice exists against the feeding of infants on sterilized milk, so that the work has not yet met with the support it deserves. Even were it true that scurvy is likely to arise from this method of feeding, it is a disease so easily prevented, so readily cured, and so very rarely fatal, that it seems liable to object to the perversion of an extremely deadly disease on a basis of a supposed increased liability to scurvy being incurred.

Remedial Treatment.—We must now consider the treatment to be adopted in a bad case of infective diarrhoea: the infant being collapsed, poisoned, and drained of fluid. The first indication is to get the child warm. It should be covered in warm blankets, surrounded by hot-water bottles and, if possible, placed upon a warm water-bed. The extremities should be wrapped in cotton-wool. If the child be extremely ill, it is best to give it a mustard-bath* at once, and as soon as it is back in bed, it should be infused, about 5 oz. of hot saline being run in under the skin of the axilla, with as little exposure to the air as possible. Next, we must try to get rid of as much as possible of the poisonous contents of the intestinal tract. The lower bowel is first washed out, and the child covered up again. Then the stomach is washed out, and when the washing is quite clear, a draught of castor oil is poured down the tube into the stomach: a feed may be given at the same time. In feeding these children, all milk must be suspended, and albumen water, lactose, and brandy given. An ounce of albumen water should be given every hour with 5, or even to drops of brandy. In addition, strychnine (up to 2 min. of a 1-400 solution) may be given under the skin.

The future treatment of the case may be conducted on some such lines as these: the gastric lavage is rather collapsing to these infants, and should not be repeated, unless necessitated by vomiting; but the rectal irrigation should be ordered to be given twice daily. Irrigation with normal saline is as useful as any other: but, if preferred, a 5 per cent solution of potassium may be used. Subcutaneous infusions should be repeated when necessary, but are not to be given more frequently than every four hours. The mustard-bath, about the strongest stimulant that we can prescribe for a baby, may be repeated when necessary. Strychnine may be given four-hourly: but it is usually not possible to give 2-minim doses at the quarter-strength solution hypodermically every four hours for more than twenty-four hours, as the infant begins to get rigid and to show twitching. At

* For this and other therapeutic measures mentioned here, see Appendix A.

the end of that time, if full doses have been given, they should be reduced by half. It is advisable to give either a small dose of castor oil (5 min. in a mixture: p. 288) or of grey powder, every four or six hours, in order to cleanse the intestinal tract. Some prefer a mixture of kassia and opium after the initial purge, but these are better not given during the first few days, unless there is abdominal pain. It is important not to allow these infants to suffer pain, as it increases the shock.

For diet, albumen water, and brandy should be given in spoon-feeds hourly, until the motions are free from curds and begin to lose their green colour. For restlessness from thirst, sips of hot water may be given between the feeds. If the case is doing well, at the end of about three days some whey may be very gradually substituted for the albumen water. The change is effected by adding an increasing amount of whey to the albumen water, keeping the feed to an ounce, so that at the end of twenty-four or forty-eight hours pure whey is being taken. If there is no set-back, the stimulants may be gradually lessened, the feeds increased in size and given at longer intervals, first 1½ oz. every 4½ hours, and then 2 oz. two-hourly. If all goes on well, a drop or two of cream may be added to the whey, and gradually increased until the child is taking about five drops in each feed. As cream is so difficult to obtain clean, and so hard to keep fresh in the hot weather, it is often preferable to add to the whey very small quantities of sterilized milk, these being very cautiously and slowly increased. Citration of the milk is here of value. At last the child is got back on to a normal diet, and as soon as possible after this, it should be removed to the country.

Particular care should be taken in endeavouring to get the child back on to milk, even in small amounts, and it must be added very cautiously. At this stage there is a great liability to a recurrence of the diarrhoea, and where this is at all of a severe type it usually ends quickly in death. Where small quantities of cream or milk give rise to renewed diarrhoea it is well to order a diet free of fat and oil until the infant's general condition is improved. For this a mixture of whey, albumen water and raw apple juice may be prescribed, to which may be added some soluble protein, such as albumin (p. 40). Lak (1) is a milk preparation which is of value in re-introducing milk into the dietary (p. 43).

It should be remembered all through the treatment of these infants, that the risk incurred by the slightest change in diet is far greater than by any attention to the medicines that are being given.

3. ACUTE ILEOCOLITIS

This is a form of acute diarrhoea, occurring most commonly in the summer season, characterized by the passage of blood and mucus in the stools. It may be accompanied by the symptoms of loss of fluid and collapse as seen in "*cholera infantum*." In London it is not a very

common condition, but it appears to be the form in which epidemic diarrhoea is found in America. It is probably due to a dysentery bacillus of the Flexner type. Occasionally it occurs in older children.

Symptomatology.—The symptoms may be of a varying degree of severity, but the cases classified under this heading in England are of the severe type. The onset is sudden, or preceded by a few days of mild diarrhoea. The temperature is raised. By the bowel is passed bright blood, mucus, and a little green slimy or watery fecal material. Considerable quantities of blood may be passed in exceptional cases. There is usually some vomiting. The amount of collapse may be extreme, and the appearance of the child is much the same as that described in the preceding article. The condition may be as acute and as grave as that produced by an intussusception; there may be, in fact, considerable difficulty in making certain that the symptoms are not due to that disease.

Morbid Anatomy.—Usually the lower part of the ileum is involved with the colon. The mucosa-membrane is inflamed, swollen, and injected, and the large bowel may show many follicular ulcers. The Peyer's patches of the ileum are swollen but not ulcerated.

Diagnosis.—As a rule the prominent symptoms of the passage of blood and mucus per rectum suggests the possibility of intussusception, and there may be little in the history of the case or in the appearance of the child to help to differentiate these two diseases. The most important points in the diagnosis are two in number. Firstly, in ileocolitis there is no obstruction, the material passed by the bowel contains fecal matter, or the finger introduced into the rectum is withdrawn colored with the green contents of the bowel; in intussusception total obstruction is present in the great majority of cases after the first few hours. Secondly, in intussusception there is usually a tumour palpable through the abdomen or per rectum. As neither of these distinctions can be said to be absolute for all cases, it must happen that sometimes the diagnosis is a matter of doubt. In such an order to facilitate abdominal palpation, an anæsthetic should be given when the tumour can be felt in nearly all cases of intussusception. This must not be shirked if there is a reasonable doubt as to the nature of the case, for the risks of leaving an intussusception undiagnosed are too great.

A rectal polyp gives rise to the passage of blood and mucus, but is usually easily distinguished by the character of the illness and the examination of the rectum. Abdominal purpura (Section IX) may lead to mistakes.

Very severe attacks of abdominal pain, causing much prostration, are in favour of intussusception or purpura rather than of acute ileocolitis.

Prognosis.—The outlook in these severe cases is bad, and the percentage of deaths is high. In mild cases, at the end of a week or a fortnight the infant is out of danger.

Treatment.—To a large extent the treatment here should be on the lines advocated for epidemic diarrhoea, to which the reader is referred (p. 261). Two differences should be noticed. After the initial dose of castor oil, it is preferable here to discontinue aperients and to give a mixture of tannin and opium in order to try and prevent excessive peristalsis causing further loss of blood. Secondly, rectal irrigation should only be practised if it does not cause any serious haemorrhage, and the irrigating fluid may with advantage contain some 3 per cent of pectargol.

CHRONIC DIARRHOEA

In infants, chronic diarrhoea is a condition met with chiefly in the children of the poorer classes, who are ill-washed and ill-fed. The mother is out at work during the day and the child is left to the tender mercies of a neighbour or in charge of some small sister. The milk is bad, the surroundings worse. In addition, various tit-bits are given from time to time, pieces of banana, pickled salmon, cucumber, currants, sweets—anything that appears tasty. In this way many slight attacks of diarrhoea are set up, and gradually the condition becomes chronic. Amongst the better classes, chronic diarrhoea is much less common, and is usually attributable to food which is unsuitable for the child. Prematurity, rickets, syphilis, tuberculosis, or any condition that may produce wasting, will conduce to chronic diarrhoea. Sometimes the condition appears to arise during convalescence from measles or the other fevers of childhood. Tuberculous enteritis is, it should be noted, a comparatively rare cause under the age of eighteen months.

In older children, chronic diarrhoea is not a common symptom apart from tuberculous enteritis, and in any persistent case this disease should be suspected. An undiscovered or unsuccessfully treated empyema may give rise to amyloid changes in the intestine with consequent diarrhoea. Apart, however, from such serious organic diseases as these, a form of diarrhoea is attacks so frequent as to be almost a chronic state, may be found in children who are improperly and irregularly fed, and who very often are infected with thread-worms. A third class is that described by Tromsden as *diarrhoea nervosa*. Here almost directly following a meal, there are severe griping pains and an evacuation of the bowels (tenesmic diarrhoea), the motion consisting of undigested food and mucus (p. 266). A rectal polypus may cause frequent discharge of blood and mucus, which may be called diarrhoea by the child's mother. Occasionally one meets with an idiot child, or even with one of normal mental development, who has never been taught

to take any solid food even at the age of six or seven years; in such, there is a frequent liquid evacuation containing a very small amount of solid residue, which is regarded as being due to perpetual diarrhoea.

Symptomatology.—In small babies the symptoms are well known and form the common picture of a marasmodic infant. Wasting is the most prominent of these. The infant's face is thin, the features pinched, the nasolabial folds are deep and may be double or even triple on each side of the mouth. The skin is transparent, moist, and often spotty. The abdominal wall shows the peristalsis of the coils of intestines clearly through it. The motions are passed frequently, are usually offensive and loose, and have a great tendency to become green in colour. The buttocks are covered with an excruciating rash. Lactonic diarrhoea is very commonly present. If death occurs it is usually the result of a more severe acute attack of diarrhoea such as has been described on a previous page. Oedema, purpura, head-retraction, and hyperpyrexia may be seen; often broncho-pneumonia terminates the case.

In older children few symptoms are seen apart from the diarrhoea. In serious cases there is wasting, and there may be other signs or symptoms of the disease which is causing the diarrhoea. Occasionally, even in children of eight or nine years of age, oedema may be found, and may be so prominent a symptom as to mask the real condition present.

Morbid Anatomy.—The examination of the internal organs in a case of chronic diarrhoea shows no peculiar lesions: the changes are simply those of malnutrition. The alimentary tract is wasted, the intestinal walls thin, almost transparent, and as a rule showing swelling of the follicles of the colon. Microscopically there is destruction of the glands, and atrophic and even cirrhotic changes are present. The gastric juice contains very small quantities of ferments and hydrochloric acid, and doubtless the secretory power of the glands throughout the intestine is diminished. The difficulty, therefore, in the treatment of these cases lies in the fact that both the digestive and assimilative powers of the intestinal tract are seriously deranged.

Apart from these changes there is little to note. The mesenteric glands are enlarged, but show no evidence of tuberculosis. The middle ears contain some mucopus, but there is little or no injection of the tympanic membranes. Broncho-pneumonia is often present; collapse of lung, usually only superficial, is almost inevitable. Very rarely thrombosis of the intra-cranial sinuses is found.

Diagnosis.—In infants under eighteen months, abdominal tuberculosis is not a common cause of chronic diarrhoea, and if due to this the diagnosis is usually easily made by palpating the hard large mesenteric

glands. In the absence, therefore, of definite evidence, chronic diarrhoea at this period of life should not be regarded as tuberculous. At a later age, as has already been stated, a persistent diarrhoea is very commonly due to tuberculous enteritis.

Prognosis.—In older children this depends upon the cause of the diarrhoea. In infants, the best guide to prognosis is the reaction to treatment, but even this is of great uncertainty. With a child having so slight a hold upon life, in many cases no accurate prognosis can be made. Certain signs are, however, of value as forecasting a fatal issue: these are, icterus, peripara, great abdominal distention in non-tuberculous cases, head-retraction, hyperpyrexia, and as a rule an acute attack of diarrhoea or bronchopneumonia.

Treatment.—The treatment of an emaciated infant with chronic diarrhoea demands the utmost patience and thought. On general lines it is similar to that which has been described for acute diarrhoea, and in all cases the measures there enumerated should be used when necessary. Strict cleanliness must be insisted upon—a point often forgotten—strict rest enforced. The child must be kept in its cot, and not taken up into the mother's or nurse's arms.

The great difficulty in treating these infants lies in their inability to digest milk satisfactorily; yet it must be our aim to find some form of milk which agrees with them, for until they can take milk no real progress is usually made.

Where the diarrhoea is at all severe, it must be treated on the lines set down under the heading of acute diarrhoea. Rectal irrigation with, if necessary, gastric lavage should be practised. All milk should be suspended for the time, either water, whey, veal or chicken tea being substituted for it. As the stools become less frequent and begin to lose their green colour, some cream may be added to the diet, a few drops only being given at first, with the addition of some lactose and a little salt.

Such a mixture as this is often taken well, but its use is of only temporary value, and we have to try to get the infant back on to a milk diet. There is, I think, no question that the substitutes for milk, however valuable for the time, are not satisfactory if used for more than two or three weeks.

Milk is therefore to be introduced into the food. At first a dash only may be added to the mixture, and subsequently larger proportions may gradually be attained. Peptonised or pasteurised milk may be first used; citrated milk and humanised milk are also of great value. If these are well borne the child is enabled gradually to take an ordinary milk-mixture, boiled water or lime-water being used as the diluent rather than barley-water. Citration is often of value for long periods, enabling a stronger mixture to be taken than where the diet is not rendered easily-digested by this means.

The various methods of overcoming acid- and fat-indigestion are summarized on pp. 59 to 60.

For drugs, an initial dose of castor oil may be administered, and a mixture containing five or ten drops of the oil is usually to be given regularly at first. When the stools become less offensive, some bismuth and soda, with or without opium, may be given. Preparations of chalk, catechu and other astringents may be administered. Silver nitrate is sometimes of considerable value, a twenty-fourth of a grain being given to a child of two years every four hours. Of more value in checking the diarrhoea is the rectal lavage already mentioned. This may be given with saline or with a 3 per cent solution of protargol.

In older children, apart from the tuberculous cases which are considered on page 142, regulation of the diet, rest in bed, a dose of castor oil followed by a single diarrhoea mixture, are the measures which, as a rule, rapidly cause a subsidence of the symptoms.

LIENTERIC DIARRHOEA.

By this term is meant a condition in which an action of the bowels occurs directly or soon after food is taken. It is common in young subjects and is a symptom indicating abnormally active peristalsis of the intestine set up by a normal stimulus. It exists in two groups of cases which correspond roughly with the ages of infancy and childhood.

In infants lienteric diarrhoea is very common, and is usually associated with true diarrhoea; that is, with excessive intestinal elimination. When an infant has severe diarrhoea, motions are passed frequently and at any moment; but when of a slighter type, the bowels act only when food is given. The motions passed usually contain undigested food. Here the diarrhoea should be treated on ordinary lines (by careful dieting, etc.), while opium is of considerable service in combating the excessive peristalsis. Should the symptoms continue after the motions have become more normal in character, small doses of arsenic or opium should be given three or four times daily.

In older children lienteric diarrhoea may be of the same type as that already described. More commonly it is not associated with any primary intestinal disarrangement, but is due only to an excessive peristalsis set up reflexly by food being swallowed, or seen, in some cases, smelled. In this form, although there are some griping pains and increased frequency of defecation, there is little excess of elimination. This type of lienteric diarrhoea is found in children who are, either by inheritance or acquired disease, of a nervous disposition. It is a functional nervous disorder, and is amenable to treatment on anti-neurotic lines. A quiet, ordered life, a plain non-stimulating diet, plenty of outdoor exercise, and a complete freedom from worry should be arranged. The internal administration of arsenic is a most valuable drug in allaying this symptom in these nervous children.

MARASMUS.

By some authors this is described as a disease rather than as a symptom, but this is certainly not clinically convenient, for it is apt to emphasize those cases in which the cause of the condition is obscure, at the expense of the majority in which it must be our first aim to prevent or to treat the factor which is responsible for its appearance.

Etiology.—Marasmus is seen chiefly in the infants of the poorer classes. Poverty covers a multitude of conditions—over-crowding, dirt, poor food and worse feeding—all of which are powerful factors in producing marasmus. Perpetrators and maintainers are responsible for some cases. In many more, however, the marasmus is secondary to bad feeding acting in conjunction with bad hygienic surroundings. Not seldom the child has thrived on the breast for a month or two, and has then been weaned in order that the mother may go out to work, and from that time has gradually wasted. The trusting of mothers who should be nursing is one of the great evils responsible for marasmus.

In other cases marasmus is due to inherited syphilis, or to such acquired conditions as tuberculosis or a latent empyema.

In a comparatively small group of cases—those to which the term of marasmus as a disease may be applied—the lack of progress seems due entirely to defective assimilative power, usually congenital, sometimes acquired.

Treatment.—The feeding of marasmic infants is described on pp. 45 to 61.

MELÆNA.

The passage of blood by the bowel is a common event during infancy and childhood.

In new-born infants (*melæna neonatorum*) the hemorrhage from the bowel is usually associated with bleeding elsewhere. In most cases, but not in all, it is due to a general septic infection.

The commonest cause of a small quantity of blood in the stools is mechanical injury to the bowel due to the passage of æchia in cases of constipation. Occurring after a purge as much as a drachm or so of blood may be passed.

Protrusion of the rectum, anal fissure, or hemorrhoid may be the source of the blood, while a less frequent cause is a rectal polyp. A careful examination for this should be made in all cases of persistent bleeding.

In cases of diarrhea in infants small quantities of blood are occasionally found, but not often apart from the condition of discolored or infantile scurvy, one of the less common sources of bleeding is

the bowel. In typhoid fever, in some cases of septicaemia and malignant endocarditis, melanæ occur.

It remains to mention three conditions which closely simulate each other, and are characterized by severe abdominal pain, collapse, and the passage of blood and mucus by the bowel. These are intussusception, acute hepatitis, and the "abdominal purpura" or Henoch's purpura. The differential diagnosis of these, often a matter of great difficulty, is given on page 534.

APPENDICITIS.

Acute Appendicitis.—It is necessary here to allude only to one or two points of interest about acute appendicitis as it occurs in children.

It has been reported as early as the sixth week of life, but it is very uncommon during infancy. As the age increases the liability to the disease becomes greater, until at the age of from eight to twelve years the disease is by no means infrequent. In rare instances the inflammatory changes appear to originate from the presence of threadworms within the appendix.

At the onset of the attack diarrhoea is a frequent symptom and is much more common than in adults. Not uncommonly the onset is insidious and the local signs ill-marked. The high position of the cæcum and appendix in some children must be borne in mind.

In children the course of the disease is rapid and unfavourable, the patient showing a poor resistance to the infection. Left to itself, appendicitis in children is usually rapidly fatal, and no expectations of resolution can reasonably be entertained in any but the very slightest attacks. The prognosis is therefore worse than in adults. Even where a localized abscess forms and is opened, the death-rate is higher than in older subjects.

For these reasons delay in operating is even more dangerous in children than in adults. A surgical opinion should therefore be obtained at the earliest opportunity in all cases, and subject to this, the physician will wisely urge immediate operation.

Chronic Appendicitis.—This is an uncommon condition in children, but one which is very liable to be overlooked. It gives rise to repeated attacks of abdominal pain, with vomiting, constipation, and perhaps acid intoxication. By careful examination of the appendicular region, it has to be distinguished from recurrent bilious attacks, cyclical vomiting (p. 53), and diseases of Meckel's diverticulum (p. 105).

INTUSSUSCEPTION.

Acute intussusception has many points of interest to the physician and general practitioner which require mention here. Chronic intussusception is a condition of very great rarity. Agonal intussusceptions are frequently found in autopsies upon children, but are of no clinical interest.

Etiology. Some interesting facts have been summarized by Mr. D. C. L. Fitzwilliams on this subject.* The *sex-incidence* is remarkable, the disease being thrice as common in males as in females. In which connection it may be noted that intussusception is practically confined to fat and strong children. The *age-incidence* shows that 70 per cent of the cases in children under twelve years of age arise in the first year of life. The monthly incidence during the first year is seen in the accompanying table founded upon Mr. Fitzwilliams' figures, and shows that from the fourth to the seventh month inclusive is the most



FIG. 56.—THE MONTHLY INCIDENCE OF ACUTE INTUSSUSCEPTION DURING THE FIRST YEAR OF LIFE. (After Fitzwilliams.)

nothing organic can be found to account for the origin of the disease? According to Mr. Fitzwilliams there are two factors in the production of such cases—imperfect intestinal co-ordination, and a diietetic factor. The *age-incidence*, as shown in the preceding table, supports this view. The disease becomes common when the periods of inco-ordinate movements of the intestine and of diietetic changes overlap: for it is during the beginning of the feeding period that unorthodox additions are made to the diet, to say nothing of the purges and powders that are so commonly used at this age. That wasted children are practically

dangerous period of the first year of life, 64 per cent of the cases arising during these four months.

The *seasonal incidence* is of interest in that it shows that there is no increase in the number of cases of intussusception during those months in which diarrhoea is particularly prevalent. The disease is most frequent during December and the spring.

The actual cause of an intussusception is in a few cases definitely toxicæmic. The process of invagination may be started by an injury, polypus, mesenteric band, Meckel's diverticulum, or a hæmorrhage into the intestinal submucosa, as in Henrich's purpura. Such cases as these, however, form only a small minority of intussusceptions.

To what then may we ascribe the ordinary case, in which

* *Lancet*, vol. i, 1908.

never affected may be due to the fact that they are more carefully fed, which may also explain why the disease is more common in boys than in girls.

In many cases careful inquiry produces evidence pointing towards the origin of the disease being due to some dietary error or to the administration of a purge.

The classification and varieties of intussusception need not be dealt with here.

Symptomatology.—At its onset the disease may escape recognition owing to the transient nature of its earliest symptoms. Nevertheless if carefully considered these are often very characteristic.

A previously healthy infant is seized with a paroxysm of very severe abdominal pain accompanied by great collapse, pallor and prostration. It vomits and passes a motion containing some blood. In older children the pain may be referred to the umbilicus. As the paroxysm of pain passes off, the child regains its colour and may appear quite well, sitting up and playing with its toys. Probably at this stage the invagination is not permanently formed.

Within a few hours the intussusception becomes persistent, the pain and collapse continuous, and symptoms of intestinal obstruction develop. The child is pale and cold, the temperature subnormal, the face drawn and anxious. The vomiting is now persistent, and blood and mucus are passed by the bowel in considerable quantities. Obstruction being present in nearly all cases, no fecal material is passed in the motions. Non-obstructive cases are extremely rare, particularly in infancy; they are usually due to an invagination of Meckel's diverticulum.

Still later the symptoms of acute peritonitis develop. Stercoraceous vomiting may be present after the second day, but is rare in infants.

Physical Signs.—The characteristic sign of intussusception is the sausage-shaped tumour felt through the abdominal wall or per rectum. It may be found under the liver, spleen, or elsewhere in the course of the colon. By means of a finger in the rectum it may be felt manually. The apex of the intussusception may sometimes be actually touched, when to the finger it resembles the os uteri. It may protrude from the anus. The right iliac fossa may be felt to be unduly empty. Later in the disease there is generalized abdominal distention from obstruction or peritonitis.

Diagnosis.—No risk must be run of mistaking an intussusception, for without treatment the disease is almost necessarily fatal. The symptoms are usually highly suggestive, and the abdominal tumour can generally be felt. If the tumour is small and hidden under the liver, and the abdominal wall is tight from the screaming efforts of the patient, an anæsthetic may be necessary in order that the abdomen

may be properly examined. In a few cases, even with an anæsthetic, no tumour can be felt, and here the abdomen should be opened if the symptoms point to intussusception.

From Intestinal Colic, intussusception may be distinguished by the severity of the pain and collapse, and by the passage of considerable quantities of blood.

From Acute Enterocolitis, intussusception in its earlier stages is distinguished by the severity and paroxysmal character of the pain and by the apparent return to health during the painless intervals. Later, the superintention of intestinal obstruction, with the consequent absence of fecal matter in the bowel discharges, points to intussusception. In only exceptional cases is the amount of blood passed in fecoliths comparable to that in intussusception. The palpation of the tumour is diagnostic here of intussusception.

From Henoch's Purpura, with severe abdominal symptoms, the differentiation of intussusception may be extremely difficult. In the purpuric cases there is usually no obstruction, and here the diagnosis is comparatively easy. But it must be remembered that in Henoch's purpura the bleeding into the submucous coat of the intestine may produce not only obstruction but a palpable tumour, while it may even originate an intussusception. The diagnosis may be suggested by previous attacks of purpura, or by the presence of a rash; but evidently, where obstruction and a palpable tumour are present, intussusception due to purpura cannot be satisfactorily excluded without an operation.

An Invagination of Meckel's Diverticulum may be suspected from the signs given on p. 305. The differentiation between this and an ordinary intussusception is not often a matter of great moment, in that the symptoms usually point clearly to the necessity for a laparotomy. The diverticulum alone, however, may be invaginated, and so give rise to a non-obstructive form of intussusception. It is useful to remember that acute diseases of Meckel's diverticulum are very rare in children under two years of age.

An intussusception protruding from the anus has been mistaken for a simple prolapse, polyp, or even hæmorrhoids.

Prognosis.—For all practical purposes the disease is fatal unless relieved by operation. Although it must be granted that spontaneous reductions of the invagination have occurred, they are extremely rare. The most important factors in prognosis are the age of the child and the time which has elapsed before operation is undertaken. The older the child the better the outlook. If operated upon within twenty-four hours recovery is the rule; by the second day the chances are much diminished, while by the third day the possibility of recovery is very small. In an infant, where section of the bowel is necessary, death is almost inevitable.

Treatment.—It is only necessary here to emphasise that operation

must be undertaken without any delay, and that an open operation is absolutely necessary. Any method of treatment in which the condition of the bowel is not carefully scrutinized is to be unhesitatingly condemned as unsafe. The danger of imperfect resection, of damage to the intestinal wall, or of leaving within the abdomen gas which cannot recover, is far too great to allow of any such procedure.

Spinal anesthesia has been shown to be particularly suitable for operations for intussusception (Gray).

VI.—DISEASES OF MECKEL'S DIVERTICULUM.

A persistent diverticulum is present in at least 2 per cent. of persons, and in many, probably in a majority, of these gives rise to symptoms at some period of life. Not uncommonly it causes death. It has even been said to be "a greater menace to life than an appendix vermiformis" (Porter).

Its diseases are mainly of surgical interest, and here I shall only deal with such clinical points as are suggestive of the diverticulum being the cause of symptoms. The material for this account is drawn from the writings of Mr. Tyrrell Gray.*

The diseases of, or due to, the diverticulum may be classified as follows:—(1) Inflammation of various types, as an appendicitis; (2) Affections due to acquired malpositions, such as torsion, strangulation, etc.; (3) Intestinal obstruction due to the persistence of omphalo-mesenteric remains; (4) Various forms of enterocysts, fistula at the umbilicus, etc., liable to excite the serious conditions given under the previous headings.

The presence of a persistent diverticulum may be suggested by some abdominal condition of the umbilicus, which may show indurating or may be markedly inflamed.

A diverticulum causes a tendency to repeated attacks of abdominal pain, with vomiting, constipation, and perhaps melena. Such a history may suggest in a case of acute obstruction in a child that a diverticulum is causing the condition.

It is noteworthy that a diverticulum does not give rise to such acute conditions in children under the age of two years, at a time, that is, when intussusception is so common.

Symptoms suggesting those of an intussusception without absolute intestinal obstruction may be produced by an invagination of the diverticulum, which may exist without elaborating the lumen of the gut.

In cases of acute obstruction due to a Meckel's diverticulum, there

**B. M. J.*, vol. ii, 1907, *Journal of Surgery*, 1908.

may be a rounded swelling, dull on percussion, situated centrally in the abdomen above or below the umbilicus. In the latter position it resembles to the touch a distended bladder, but is not removed by the use of a catheter.

These are some of the chief diagnostic points in connection with diseases of the diverticulum. Were it possible to recognize the origin of the slightest abdominal spasm, it would be highly advisable to have the diverticulum removed, so that the more serious attacks might be prevented.

VII.—INTESTINAL PARASITES.

OXYURIS VERMICULARIS (*Thread-worm*)

This, the commonest form of worm found in children, resembles a piece of white thread, the female being about one-third of an inch in length, and the male considerably smaller. The female has a tapering tail, while that of the male is blunter and curled. The ova are minute, round or oval bodies. According to Leuckart the ova cannot develop unless they pass through the alimentary tract, but this may readily be the case with children who are allowed to scratch themselves to relieve the irritation round the anus. Thread-worms may inhabit the whole of the large intestine from the caecum downwards.

Symptomatology.—Itching caused by the migration of the worms from the rectum during the night is the chief symptom. It comes on as a rule soon after the child has gone to bed, and may be most severe. It may cause frequency of micturition and nocturnal enuresis. The scratching may set up vulvitis or vaginitis in female children, and may be the origin of a habit of masturbation in either sex. From the associated colitis there is usually an excess of mucus in the stools.

It is important to recognize that in most cases the presence of thread-worms is a sign rather than a cause of ill-health, apart from the local conditions named. As a rule lassitude, irritability, capriciousness of the appetite, coated tongue, and, most commonly, constipation are present. Acute appendicitis is occasionally due to thread-worms.

Lastly, we come to the question of associated nervous symptoms which, from convulsions to head-rolling, have in times past been held to be due to the presence of worms. It is hardly possible to regard the parasites themselves as producing the long list of complaints with which they were formerly associated, and the association may be explained in two ways. The intestinal derangement and general bad health, so often the cause rather than the result of the presence of the parasites, are such as are likely to predispose to nervous symptoms. We are familiar with such in children of this type, although free from

worms. On the other hand, it is only reasonable to assume that the intolerable itching and discomfort which thread-worms induce may tend to increase the irritability of an already sensitive nervous system, and that such an influence may help in the development of nervous symptoms, or tend to keep them from disappearing.

Treatment.—This consists of local and general treatment, both of which are of importance.

Local treatment must be directed towards scrupulous cleanliness, the prevention of the migration of the worms, and the killing off of such as are infecting the lower part of the rectum. The parts about the anus should be bathed with a solution of perchloride of mercury (1-10,000) twice a day, and after each action of the bowels. At night the anal region should be smeared with a dilute white precipitate ointment (half the *D.P.* strength). This will prevent the migration of the parasites, and so tend to stop the itching. Rectal injections are of value in some cases but are not necessary in all. They should always be regarded as merely supplementary to the general measures mentioned above. Injectants of salt (two drachms to the pint) or of infusion of quassa may be used, but probably the best is the solution of perchloride of mercury. This last should not be injected more frequently than every third night, its place being taken on the intervening nights by the salt solution.

The general treatment consists in seeing that the child obtains plenty of fresh air in order to stimulate the appetite, and in taking measures to put the alimentary tract in order. An initial dose of calomel should be given, and this should be followed by the regular administration of an aperient. A rhubarb and gentian mixture is usually of great benefit, or grey powder, or rhubarb and soda powders may be given. Such a course of drug treatment is necessary in order to sweep away the worms infecting the upper part of the large bowel, to get rid of the mucus, and to put the intestinal tract in as healthy a condition as possible. It is the most important part of the treatment.

The application of the ointment to the anus may well be continued for some time in order to prevent the child re-infecting itself.

ASCARIS LUMBRICOIDES (*Round-worm*).

This is the next most common form of intestinal worm found in children. It is in appearance like an earth-worm, but paler and more slender. The male is about six inches or rather less, and the female about twelve inches in length. The latter is frequently found with processes protruding from its ventral surface. These are the ovaries extruded as the result of pressure. The ova are oval in shape and about $\frac{1}{16}$ inch in diameter. The worm infects the small bowel and is *fairly* solitary. Usually some number under a dozen is present, but there may be ten or even hundreds of them. Their

life-history is not altogether understood as yet, but it is probable from Eptitrit's experiments that the ova on being swallowed can develop in the intestine into the adult parasite, and that no intermediate host is necessary. They may be taken as with unwashed vegetables or impure water.

Symptomatology.—Symptoms are often entirely absent or there may be the slight ones of loss of appetite, restlessness, and irritability. Frequently colic and abdominal distention are present, and with constipation, tenderness, and perhaps vomiting, may give rise to a suspicion of acute appendicitis. The blood shows an increase in the eosinophile cells, as in other parasitic conditions.

Occasionally such acute alarming symptoms present themselves, which some have accounted for by supposing an absorption of a poison secreted by the worms. The child is seized with violent abdominal pain, becomes almost unconscious and much collapsed, and may be delirious, rigid, or even convulsed. There is much vomiting. The bowels are as a rule confined. The urine may be full of pus. In such cases the symptoms, which usually hemiplegia quickly disappear with the expulsion of the worms.

In addition, symptoms may be present from the migration of these worms. They are not uncommonly vomited and occasionally are extruded from the nose or ears. They may pass into the common bile duct and produce jaundice. They have produced fatal asphyxia from their presence in the larynx. They can perforate even a healthy intestine, but more often pass through an ulcerated portion. With the exception of the migrations to the stomach, the complications enumerated are remarkably uncommon.

These parasites very commonly escape from the bowels of children who are within a few days of death from any disease, and this fact probably accounts for the rarity with which round-worms are found at autopsies.

Diagnosis.—The passage of one round-worm suggests the presence of others. Ova found in the stools indicate that there are parasites present in the intestine. In doubtful cases smears of the intestinal evacuations should be examined microscopically.

Treatment.—Santonin is the best drug to use. An aperient should be given over night, and the santonin as a powder the next morning. Three grains with some sugar is the dose for a child of five years, and may be given in one powder or in divided doses; for a child of two years a grain and a half may be ordered. The santonin may be given with some calomel (gr. ½) or other purge, and sugar.

CESTODES (*Tape worms*).

This variety of parasite is far less commonly found than the

threads or round-worm. In children it is the beef tape-worm (*Taenia solium*) that is usually found; the pork tape-worm is of great rarity. In infants the *Taenia crassiceps*, whose larva inhabits the lice of dogs and cats, is said to be the commonest form. *Rochelecephalus latius*, the fish tape-worm, is very rare in this country.

The *T. solium* is from twelve to twenty feet in length. The segments, when fully grown, are about half an inch in length and half as wide, and the uterus is closely subdivided. The head shows four suckers, but no hooklets. The *T. crassiceps* is shorter (up to ten feet), the segments more square in shape, the uterus less closely subdivided, and the head shows in addition to the four suckers a proboscis surrounded by about twenty-six hooklets.

Tape-worms require from ten to twelve weeks for their growth, so that should all the worms except the head be expelled, this period may elapse before any more segments are passed. The segments are expelled singly or in short chains, but as the worm grows by the addition of segments at the head end, its length is not diminished. The ova are contained in the mature segments (proglottides), and are thus distributed. When swallowed by the alternate host, the shell is dissolved by the gastric juice and the embryo set free. The hooklets with which this is provided enables the embryo to pass through the stomach-wall and to migrate to any part of the body of its host, where it becomes changed into a vesicle, a cysticercus. The cysticercus of *T. solium* is occasionally found in man. By eating the raw or partially cooked flesh of the infected animal (beef, pork or fish), man becomes infected. Passing into the stomach the cysticercus loses its wall, and the head of the worm contained in it passes into the intestine, and there develops into the mature parasite.

Symptomatology.—A tape-worm may cause no constitutional symptoms at all; but if the child is in an indifferent state of health indefinite symptoms, such as coated tongue, a capricious appetite, sometimes poor, sometimes excessive, slight attacks of colic and of diarrhoea may be present. Great wasting and anaemia are not as a rule seen in cases of infection with the beef or pork tape-worms.

Diagnosis.—Faint segments of the parasite are passed, its presence cannot be diagnosed. Unless the head has been expelled, return of the parasite is almost assured.

Treatment.—Indigestion may be prevented by the proper cooking of meat, so as to destroy the cysticercus. Although there is less danger in eating improperly cooked beef than pork or sausages, yet it must be mentioned that infection has originated from the administration of raw meat juice to children.

The usual method of treatment is as follows:—For two or three days aperients and alkalis are given in order to empty the intestines and

get rid of any excess of mucus that may be present. At night a very light meal is given, preferably only milk, and a dose of castor-oil is administered. In the morning the child has a saline aperient, but no breakfast. He should be kept in bed. When the bowels have been freely opened, the liquid extract of male fern is given. For a child of eight or ten years old, a drachm or more may be given either in one dose or in four doses at hourly intervals. In a mixture the drug is made up with spirit of cinnamon, syrup, and mucilage of acacia to an ounce, thus:—

R. Lix. Ficus 1ij	℥i	Syrup	℥ss
Sp. Cinnamon	℞ssij	Mucilag. Acacia	ad ℥j

For a child of two years half these doses may be ordered. Two hours later, half an ounce of castor oil should be administered. The motions passed must be examined carefully for the head. If unsuccessful, the worm breaks at the neck. Should this be the case, no further treatment should be given until segments are again passed, which will occur after an interval of about three months.

There are not a few failures with this line of treatment, even when most carefully followed, and sometimes thyroid will succeed in dislodging the head where male fern has failed. Ten grains or more of the drug may be given to a child of ten or twelve years old in one dose, it being preceded and followed by aperients. There is no danger in such large doses if no alcohol is given, but as thyroid is soluble in alcohol, serious symptoms may arise should this be present in the intestine. The amount of fat in the diet should be diminished during the treatment by thyroid.

VIII.—DISEASES OF THE PERITONEUM.

ASCITES.

In children, as in adults, ascites may be due to diseases of the heart, lungs, or kidneys. Apart from these conditions, ascites is most commonly due to tuberculous peritonitis. Carcinoma of the liver, usually syphilitic, is responsible for a smaller number of cases. A much rarer cause is enlargement of the glands in the hilum of the liver, such as may be due to tuberculous, malignant disease, or acute lymphatic leucæmia.

In some cases no cause for the ascites can be found. Probably, however, in the majority of such instances it is due to tuberculous peritonitis.

Chylous ascites has been reported in children, and is usually associated with abdominal tuberculosis.

Diagnosis.—In well-marked cases, ascites is easily recognized. Such sources of error as hydromphrosis, ovarian or hydatid cysts, are uncommon in children.

There may be considerable difficulty in recognizing a very small effusion and in differentiating between early tuberculous peritonitis, the protuberant abdomen of rickets, and the distended abdomen of ascites uræmic. It is well to remember that in rickets the liver is commonly enlarged, and owing to the laxity of its connections and supports may be movable, so that it may produce shifting dullness in the right flank simulating a very small collection of fluid.

Treatment.—The treatment of ascites is that of the disease causing it. Where the fluid is sufficient to give rise to bad effects upon the heart or respiration, it may be removed slowly by paracentesis. The treatment of tuberculous peritonitis is given on p. 142.

PERITONITIS.

The following types may be considered: (1) Acute purulent peritonitis; (2) Chronic non-tuberculous peritonitis; (3) Tuberculous peritonitis.

1. **Acute Purulent Peritonitis.**—In the new-born this is usually a sequel to an umbilical infection. In older children it may result from intussusception, appendicitis, vulvo-vaginitis, and other less common conditions.

Pneumococcal Peritonitis is described fully under the pneumococcal infection (p. 133).

Rheumatic Peritonitis has been ascribed to by some authors on the grounds, as a rule, that there has been a chill preceding the attack, and that no other cause for the peritonitis could be recognized. In view of the modern position of rheumatism amongst the bacterial infections, these cases can no longer be admitted as properly rheumatic. Excepting those areas of exudate seen in fatal cases of rheumatic pericarditis on the upper surface of the liver (and those probably result more from oedema than from inflammation) I have never seen any peritonitis which might truly be considered a part of an infection by the rheumatic organism.

2. **Chronic Non-tuberculous Peritonitis.**—In the new-born, peritonitis of this form has been ascribed to inherited syphilis and associated with the production of adhesions which, if originating during fetal life, may result in congenital stenosis of the intestine. Little, however, is as yet known with certainty about this form of peritonitis, whose existence is even doubtful.

In older children a form of chronic peritonitis with ascites is occasionally met with which is apparently not due to tuberculous. It simulates, however, in nearly all respects the tuberculous form, but is said to be associated with less fever and wasting. Its origin is quite

obscure. It is not unlikely that most of such cases are in reality due to a slight tuberculous infection.

3. **Tuberculous Peritonitis.**—This is described under tuberculous (p. 120).

IX.—DISEASES OF THE LIVER.

In children the liver is relatively larger than in adults. Its upper border is slightly higher in young than in old subjects, and reaches the fifth, seventh and ninth spaces in the mammary, mid-axillary, and axillary lines respectively. In infants its lower level can be felt just below the costal margin, but in older children it does not project from under the chest-wall.

At birth the liver weighs 4½ oz. (Birch-Hirschfeld), at one year 11 oz., at two years 14 oz., and at three years 19 oz. (Holt).

DISPLACEMENT.

In wasted children the liver tends to drop owing to the weakness of its supports. In rickets, falling-in of the chest-wall is an additional cause of downward displacement of the organ. In cases of right pleural effusion the liver may be pushed downwards. With ascites, large abdominal tumours, or great abdominal distention, it may be displaced upwards. In transposition of the viscera the liver may be fixed on the left side.

MALFORMATIONS.

The only malformation calling for attention is one of the bile-passages.

CONGENITAL OBSTRUCTION OF BILE-DUCTS.

Etiology.—The origin of this rare condition is very obscure. It has been regarded as a primary maldevelopment (Thomson), or as due to a descending cholangitis causing cirrhosis with secondary obliteration of the ducts (Rolleston). On the latter view it has been termed "congenital hepatic cirrhosis with obliteration of the bile-ducts," and must be regarded as due to some unknown toxin. In exceptional cases it is probably due to inherited syphilis.

The condition shows no noteworthy sex-incidence. More than one instance may occur in one family.

Morbid Anatomy.—Any part of the biliary passages may be affected. Most frequently the common bile-duct is imperforate in such

or little of its course. It may be represented only by a fibrous cord. The ducts may all be absent. Obliteration of the cystic duct alone has been recorded. The gall-bladder may be absent, but is usually present, although small and rudimentary. It may be distended. The liver is enlarged, hard, and dark green in colour. It shows much sinusoidal and multilobular cirrhosis. The bile capillaries are dilated. The spleen is enlarged, and may show an excess of fibrous tissue. Fibrosis of the pancreas has been reported (Enomoto).

Symptomatology.—*Jaundice* is the chief symptom. It may be present at birth, and in nearly all cases it develops by the end of the first week of life. It has been recorded as delayed until the third week. It becomes intense, and at a late stage may be associated with hæmorrhage into the skin, from the umbilicus, nose, or elsewhere.

The stools are free from bile-pigment throughout life, except when the cystic duct alone is obliterated. The motions are white and chalky. The urine is deeply bile-stained, but contains no urobilin. This latter fact is proof that the biliary obstruction is complete, since for the production of urobilin (formed by the action of bacteria upon bilirubin) the presence of bile in the intestine is essential.

The liver is usually enlarged to 2 or 3 inches below the costal margin. It may reach to the level of the umbilicus or even lower. It is hard and regular, but late in the disease it may contract slightly. The spleen is enlarged downward about 1 inch and may be indistinctly firm. The abdomen is distended, the superficial veins may be dilated. Ascites may develop. The general nutrition of the child suffers severely sooner or later. It may remain fat for some weeks or even months.

Diagnosis.—The condition is recognized by the presence of complete biliary obstruction from birth. This is shown by the absence of bile-pigment in the stools, and of urobilin in the urine.

Prognosis.—Death is inevitable. It may occur within the first two months, but frequently it is delayed until between the sixth and twelfth months. One case survived for fifteen months. Death is due to emaciation, hæmorrhage, vomiting, or broncho-pneumonia.

Treatment.—There is no treatment capable of influencing the course of these cases.

JAUNDICE.

It is convenient to consider jaundice as it occurs in the new-born and at later age separately.

1.—ICTERUS NEONATORUM.

This is of two varieties, the physiological and the pathological.

Physiological Jaundice.—This is seen in about one-third of new-born infants. It usually appears during the first week, most commonly from the third to fifth days, and remains according to its severity from three to fourteen days. The depth of the coloration of the skin is variable, and tends to be more marked in weakly or premature infants than in the strong. The stools are mottled in colour, and the urine contains bile-pigment in the more intense cases only.

The cause of this form of jaundice has been much discussed, but is not yet known with certainty. It is probably concerned with the destruction of red blood-cells which occurs directly after birth.

The clinical significance of physiological jaundice is very small. It does no harm and causes no excessive loss of weight during the first week of life. Although it tends to be marked in the delicate and premature, such deaths as occur amongst them cannot be attributed to the jaundice.

The distinguishing points between the physiological and pathological types of *icterus neonatorum* are mentioned under the latter condition.

Physiological jaundice requires no treatment.

Pathological Jaundice.—(*Icterus Neonatorum Gravis*).—This most serious condition is usually due to a pyogenic infection. In most cases the organism gains entrance through the umbilical vessels, but occasionally the eyes or air-passages give admittance to the infecting agent. The infection may enter through the umbilicus before or after the separation of the cord. Usually there is redness round the umbilicus, often hemorrhage or a purulent discharge from the stump of the cord. Local signs may be absent during life, but even here the infection post mortem can be traced to the umbilicus as a rule, for the vessels are found to be thrombosed and to contain pus. The arteries may be more affected than the veins. The liver is congested, and may show thrombosis of the portal vessels and multiple abscesses.

Much rarer causes are congenital obliteration of the bile-ducts, syphilitic cirrhosis of the liver, and biliary calculi. These are described elsewhere, and need not here be referred to further.

Symptomatology.—The jaundice may be slight, but in severe cases is usually very well marked. The stools are not wholly devoid of colour. Diarrhoea is usually present. The temperature is raised and the infant rapidly wastes. Hemorrhages are often present. They occur most frequently in the subcutaneous tissues, from the umbilicus or from the bowel. In some cases the symptoms are pyæmic rather than septicæmic and peritonitis, meningitis, and arthritis may occur. The last is said to be most common in gonococcal infections.

Diagnosis.—The severe cases are differentiated from physiological jaundice by the presence of some local signs of sepsis at the umbilicus,

or, where these are absent, by the character of the symptoms. The extreme illness of the infant, the fever, diarrhoea, hæmorrhages, and emaciation usually render the diagnosis easy.

Those cases due to congenital obstructions of the bile-ducts are differentiated from the infective class by the absence of pigment in the stool, and of urobilin in the urine (p. 313).

Prognosis.—The outlook is extremely bad, only a very few of these infants surviving. Death may be due to emaciation, bronchopneumonia, diarrhoea, convulsions, hæmorrhage, or pyæmic abscesses.

Treatment.—Prophylaxis is of most importance. The septic cases are no longer preventable as puerperal fever, as is shown by the fact that in modern maternity hospitals they are no longer seen.

When the infection has occurred the treatment is partly local and partly general. If there are signs of suppuration at the umbilicus, fomentations should be applied. Where there is erysipelas an ointment containing ichthylol (10 to 20 per cent) may be of benefit. The general treatment consists in supporting the infant's small amount of strength, and in alleviating symptoms as they arise.

2.—JAUNDICE IN OLDER CHILDREN.

Catarrhal jaundice, associated with gastro-duodenitis, is by far the most common form of jaundice in children. The next most common causes are severe right heart failure and syphilitic cirrhosis of the liver. It is sometimes seen in pneumonia, purulent pericarditis, empyema, in septicæmic infections, and in acid intoxication. Jaundice may be due to the presence of a round-worm in the common bile-duct, to acute suppurative cholecystitis, and to malignant disease of the liver, or of the glands in its hilum. Cirrhosis of the liver, other than syphilitic, hydatid cysts, and biliary calculi are very rare causes of jaundice in children.

A peculiar form of jaundice occurring in several members of a family is known as congenital family cholemia. This, and catarrhal jaundice, are the only types requiring description here.

1.—CATARRHAL JAUNDICE.

As has been mentioned, this is the commonest form of jaundice in children. It does not, however, occur in infants. It is due to gastro-duodenitis, which may arise as a primary condition, or occur as a sequel to such infections as pneumonia and influenza. Certain atmospheric conditions appear to predispose towards catarrhal jaundice, so that it is seen in small epidemics.

Symptomatology.—The symptoms due to gastro-duodenitis are usually easily traced, the jaundice being preceded by two or three days

of an illness of the nature of a "bilious attack." There are often vomiting, slight fever, and some pain or tenderness in the epigastrium. The bowels may be confined, but are often relaxed, the stools being loose and offensive. The jaundice appears first in the conjunctivæ, later in the skin, and is seldom more than slight in amount. The stools are pale, fatty, and offensive, but rarely colorless. The urine is bile-stained. The tongue is thickly coated, and the child complains of headache, and feels languid and generally wretched. The liver is a little enlarged and tender. Itching of the skin and bradycardia are seldom found in the jaundice of children.

The jaundice may last for about a fortnight, and rapidly disappears when the general symptoms abate.

Diagnosis.—This, as a rule, presents no difficulty. Care must be taken in post-pneumonic cases to exclude purulent pericarditis or empyema.

Treatment.—If the temperature is raised, or there is vomiting or diarrhoea, the patient is best put to bed. In other cases he may be confined to his room. A limited diet should be given, but in children it is difficult satisfactorily to reduce the fat taken. Broths may be used to replace some of the milk in the diet. Fatty meat juices, powdered chicken, or fruit, may be given. No large amounts of fat or carbohydrate should be allowed.

At the outset of the treatment a dose of calomel should be given, and this is often wisely followed by the regular administration, two or three times daily, of small amounts of calomel, or grey powder, with some soda. Each morning a saline aperient should be given. Where the symptoms of gastro-duodenitis continue, a bismuth mixture may be of great benefit.

4.—CONGENITAL FAMILY CHOLÆMIA.

(*Recurrent Familial Jaundice; Congenital Acholia; Juvenile*).

Etiology.—The disease may appear in more than one member of a family, and in succeeding generations. It may affect either sex. It is to be regarded as a congenital and familial disease rather than as an acquired condition.

The jaundice here does not seem to originate from any primary hepatic disorder, but is probably referable to an abnormal blood-condition, which in its turn is due to a congenital defect in the blood-forming system. These points are not at present, however, settled. The reader may be referred to papers by Haskins and Dudgeon,* and by Forryton.[†]

* *Quid. J.* 1901, 1902, Jan., 1903.

† *Lancet*, Jan. 15, 1900.

Symptomatology.—The discoloration of the skin is usually present at birth, but may escape notice for the first year or two of life. The amount of persistent jaundice varies in different cases; in some only the sclerotics are permanently tinted. In most cases there are periods in which there is considerable deepening of the jaundice. The general health of the patient is good, but during the exacerbations there may be some irregular pyrexia, languor, and irritability. Such symptoms seem dependent chiefly upon the anæmic state of the patient. The stools are never acholic. The urine does not contain bile-pigment, although it looks bile-stained; it usually shows an excess of urobilin. The spleen is constantly enlarged, exceptionally to the level of the umbilicus. During the acute phases it may become tender. The liver may be slightly enlarged. It shows no anatomic changes.

The blood shows important alterations. Anæmia is present, and is increased during the periods of deepened jaundice. The red cells and the haemoglobin are below normal; the colour index is lowered. The white cells show little definite change. The blood-serum contains bile-pigment. The red corpuscles are abnormally fragile as tested by saline solutions *in vitro*. Nucleated red cells are found; microcytes are numerous.

Diagnosis.—In a typical case the disease is easily recognised. It has to be distinguished from Hanot's cirrhosis (p. 495) and primary splenomegaly.

Prognosis.—The disease is compatible with good health, and does not seem materially to shorten life. It may persist at all events until middle life.

Treatment.—Treatment of the anæmia by means of iron and arsenic seems to be of most benefit; but the disease is little influenced by any form of treatment.

CIRRHOSIS.

Cirrhosis of the liver is an uncommon disease in children. In its most definite and frequent form it is due to inherited syphilis. Alcoholic and tuberculous cases are very rare. In a few instances the cirrhotic changes appear to be originated by an attack of one of the exanthemata. Congenital cirrhosis with obliteration of the bile-ducts is described on p. 312. A similar type of congenital cirrhosis, but unaccompanied by any obstruction to the bile-ducts has been reported. Hanot's cirrhosis is considered on p. 308.

There remains a group of cases in which cirrhosis of the liver occurs without any recognisable cause. Since the introduction of the serum test for syphilis, a number of such instances have been shown to be syphilitic, and in all doubtful cases this infection should be suspected.

Symptomatology.—The symptoms do not differ from those seen in adults, and consist of anæmia, slight jaundice, epistaxis, and other hæmorrhages. Enlargement of the spleen is more constant than in older patients, and is usually well marked. Diarrhoea is often a prominent symptom. The liver is enlarged in nearly all cases, but late in the disease it may be shrunken and smaller than normal.

The syphilitic cases are described on p. 185.

Diagnosis.—Hepatic carcinoma has to be distinguished from tuberculous peritonitis, which is the commonest cause of anæmia in children, and from the various causes of jaundice.

Prognosis.—Death, as a rule, occurs within a few months of the onset of jaundice; but in older children it may be delayed for two or three years. The syphilitic cases are most amenable to treatment.

Treatment.—In doubtful cases the treatment may well be an anti-syphilitic line, while those which are due to syphilis should be treated strictly in the same way. Apart from this, and the removal of any possible cause for the disease, the treatment must be symptomatic.

HANOT'S CIRRHOSIS.

This is a very rare disease of unknown etiology, which is sometimes seen in older children, but never in infants. It is more common in boys than in girls, and may occur in more than one member of a family.

Jaundice is an early and persistent symptom. It gets slowly worse. The liver is considerably enlarged, but remains smooth, or at the most is finely uneven. It is deeply bile-stained. The cirrhosis is of a fine type, at first sublobular, later intralobular and pericellular. The spleen becomes very large. Clubbing of the fingers is a peculiar symptom of this disease. Occasionally some of the superficial lymphatic glands are slightly enlarged; post mortem some swelling of the glands in the portal tissue is commonly found. The blood shows a profound secondary anæmia. The urine contains bile.

Hanot's cirrhosis may last for many years without causing serious impairment of health. Ebbec periods, in which there are abdominal pains and deepening of the jaundice, may occur; and during these the patient feels ill. These attacks are similar to those seen iniliary cholemic jaundice. Growth is much impaired, the child remaining short and thin.

Towards the end of the disease there occur hæmorrhages into the skin or from the gums, nose, or elsewhere. Haemorrhoids is rare. Shortly before death ascites may develop, but it is not common and is always a late sign. Death may be due to chæmia or to some terminal infection.

Diagnosis.—Where a clear history is obtainable Harot's cirrhosis can be diagnosed with fair ease from family acholuric jaundice (p. 316). The early and permanent jaundice and the absence of ascites differentiate the condition from primary spherozoegaly, and from other forms of cirrhosis. Hydatid disease of the liver may give rise to a suspicion of Harot's cirrhosis, but in the latter the enlargement of the liver is uniform.

Treatment.—The disease is always fatal. Few examples of it reach the age of thirty years. General measures must be adopted to keep up the patient's strength as much as possible. Some improvement has occurred after drainage of the gall-bladder.

FATTY DEGENERATION.

Fatty changes in the liver are commonly found in autopsies in children; but although they may give rise to some enlargement of the organ, they seldom cause any severe functional derangement. Such changes are of a secondary nature and are particularly prone to develop in association with chronic diarrhoea. A fatty liver is thus present in many sickly children, and in marasmic infants. In tuberculous enteritis the changes in the liver are well marked. In fatal cases of acid intoxication (p. 85) fatty degeneration of the liver is constant, and often extreme.

MALIGNANT DISEASE.

New growths in the liver are usually secondary, but it often happens that the primary growth is not recognisable clinically, so that it is the hepatic condition which has to be diagnosed.

The growth is usually sarcomatous, and is more common in infants than in older children. In many ways, in addition to its age-incidence, this condition resembles that of renal sarcoma. Abdominal distention is usually the most marked feature. It is generally associated with ascites, and often with slight jaundice. It is noticeable that great enlargement of the liver from sarcoma may exist with a very fair general condition, another point in which the disease simulates renal sarcoma. Even within a week or two of death the child may appear surprisingly fat and well. The spleen is usually enlarged.

Diagnosis. From pythitic embolism of the liver the diagnosis may be of great difficulty. A thorough abdominal examination under an anæsthetic is necessary in many cases. In infants a condition of comparatively good nutrition is in favour of sarcoma—which is of very rapid growth in these cases—rather than of pythia. The outlook in both diseases is extremely bad, so that in infancy an error in diagnosis is not of great moment.

FUNCTIONAL DERANGEMENT (Bilious Attacks)

Cases showing evidence of functional disorder of the liver are common enough in children, but form a rather indefinite clinical group. They are for the most part associated with chronic intestinal indigestion and chronic constipation. Such children, although not appearing ill, look out of health. The complexion is sallow, the appetite poor, the tongue a little coated, and covered, perhaps, with slime. Indigestion, flatulence, and abdominal discomfort are often present. The bowels are usually confined, and the stools are pale or grey. Such cases are best treated on the lines laid down for chronic constipation.

Bilious Attacks.—In children, short illnesses, which are apt to be described as bilious attacks, are very common. The child's usual health may be fair, but he is subject to periodic attacks of vomiting, with slight fever, headache, epigastric pain, and furred tongue.

Most commonly these so-called bilious attacks arise in children who are the subject of chronic intestinal indigestion and constipation, and may be due to improper food, chill, or continuance of the bowels of more severity than usual.

On the other hand, there are more serious conditions in which similar attacks may occur, of which chronic appendicitis, migraine, and cyclical vomiting must be mentioned. In a few cases, renal disease, hydrocephalus, pyelitis, or calculus may be the origin of the attacks. Lastly, the possibility of vomiting, ascribed to "biliousness," being in reality due to the onset of acute meningitis or other intracranial disease, must be borne in mind.

RARE DISEASES OF THE LIVER.

Tuberculosis of the liver rarely gives rise to any clinical symptoms, but is described as a possible cause of cirrhosis.

Suppurative Pyelophlebitis.—In the new-born this may result from an umbilical infection. In older children it is most commonly due to appendicitis.

Amylloid Degeneration, Acute Yellow Atrophy, and Hydatid Disease, are all very rarely found in young subjects.

Biliary Calculi are extremely uncommon in children. In about half the cases, they have occurred in the new-born, and have caused persistent jaundice with a fatal termination within a month. In these, very small calculi are present in the common bile-duct, but there are no symptoms pointing to such a cause of obstruction. In older children, calculi are occasionally found, and give rise to much the same symptoms as in adults.

SECTION VI.

DISEASES OF THE RESPIRATORY SYSTEM.

COUGH.

It is well to consider at the outset of this section the significance of a cough as it occurs in childhood. The most important point to be remembered is that the commonest cause of a cough in children is some pharyngeal abnormality, and not bronchial or pulmonary disease. In the great majority of children brought to the doctor for a persistent cough there are no signs of intrathoracic disease, and enlarged tonsils or adenoids, or both, are the cause of the symptom. In our patient practice the spatula is of more use in this connection than the stethoscope. Although there are no possibilities by which this "throat-cough" can be recognized without physical examination, it is well to point out its usual characteristics. During the daytime, as a rule, it is a loud, loose cough, due to exudation running down the back of the pharynx, which is easily coughed up and then swallowed. The cough is not usually at all paroxysmal in character during the day, but is just a frequently recurring, loose, rattling cough. At night, however, it generally is more alarming; it becomes much more paroxysmal. The child on waking, gives a series of coughs until his face becomes red, even slightly cyanosed, but there is no inspiratory whoop following the expiratory efforts. The cause of this is simple. With adenoids or enlarged tonsils the child sleeps with the mouth open, the throat becomes dry, and a choking or tickling sensation sets up the severe coughing. It will be seen, then, that this cough simulates an incipient pertussis or a disappearing bronchitis.

A long urethra may cause a frequent, dry, irritable cough, at its worst when the patient lies on his back.

The cough associated with any serious pulmonary condition is accompanied by dyspnea, as is that due to heart disease with pulmonary congestion. An increased respiration rate suggests at once that the throat alone is not to blame, and that the chest must be examined. The cough associated with pleural pain is short and distressing, as in adults.

The cough of pertussis has been described (page 246), and its points of similarity to, and difference from, that due to enlarged bronchial glands or emphyse have been discussed.

Lastly, there is a nervous cough, for which no organic basis can be discovered. It is found in older children who are of nervous type, and is usually a persistent, dry, short, worrying cough. On the other hand, the loud hysterical "bark" is not uncommon. Most frequently it has its origin in some throat condition, the loud "bark" being added on the patient's own initiative, or being copied from some grown-up performer.

Treatment.—The treatment must be directed towards removing the cause of the cough or the disease from which it arises. If of purely nervous origin it is best dealt with on antineurotic lines; removing the child from worrying circumstances, ensuring plenty of fresh air, and a suitable amount of exercise. A dose of bromide may be given with benefit at night before bedtime.

HÆMOPHYSIS.

This is not a common symptom in children, and for such hæmorrhage to be of any large amount is very rare. This is not explained merely by the fact that children under five or six years do not bring up their sputum, but because severe mitral stenosis and excavating pulmonary tuberculosis are both comparatively uncommon in children under twelve.

Apart from local causes in the mouth or throat, the commonest disease associated with hæmoptysis is peritonitis.

In pulmonary tuberculosis it is a rare symptom under the age of about eight years, but it does occur very occasionally in children under four years of age, where there are in addition gangrenous changes in the lung, and may be of such severity as to be fatal. Gangrene of the lung is practically the only cause of severe hæmoptysis in infants.

Pneumonia, heart disease, and bronchiectasis are also causes of hæmoptysis. Severe blood diseases and the hæmorrhagic forms of various infections may give rise to hæmoptysis.

I.—DISEASES OF THE NOSE.

EPISTAXIS.

In the new-born, epistaxis is uncommon, and usually indicates inherited syphilis. In infancy it is one of the later manifestations of scurvy. In young children it is often due to injuries, such as blows or falls, or to simple picking of the nose. A foreign body, such as a bead, pea or boot-button, in the nose, is not an uncommon cause. A small ulcer on the septum may be the origin of the bleeding.

Care must be taken to exclude the possibility of the hæmorrhage

being from the pharynx and due to adenoid vegetations. On the other hand, the blood coming from the nose may be concealed, being swallowed and afterwards vomited.

Epistaxis may be associated with various diseases:—nasal diphtheria, whooping-cough, typhoid, the hæmorrhagic forms of the acute specific fevers, particularly measles with hæmophilia and severe anemia. It is not uncommon in heart disease in its graver forms.

It may also occur in robust children after exertion, and is often here associated with temporary constipation. It is particularly common at the age of puberty. In such it may be regarded as of very little significance. But on the other hand, epistaxis occurring in an anæmic child should cause the physician to make a careful examination of the size of the heart, for many such cases are undoubtedly examples of early rheumatism, as was pointed out some years ago by Dr. S. Phillips. There are associated pains in the limbs, headache, pallor, fretfulness, nervousness, disturbed sleep, constipation, irregular excitable pulse, and slight dilatation of the heart.

Dr. Sutherland has pointed out the association of some cases of epistaxis with cyclical albuminuria.

Treatment.—This depends upon the cause, and for most instances the treatment need not here be detailed. Where due to an ulcer on the septum, the nasal cavities should be cleaned out as well as possible, and adrenalin solution applied, or the ulcer may be touched with chromic acid. The nasal cavities may be filled with strips of gauze damped with adrenalin. Plugging of the posterior nares is so commonly followed by inflammation of the middle ear as to render it a dangerous procedure. It is never necessary in children.

Where occurring in robust children, a course of saline aperients may be given.

ACUTE NASAL CATARRH.

At all ages, from infancy onwards, children are apt to suffer from acute nasal catarrh, a predisposition to which may run throughout a family. There are many factors which tend to bring on these attacks. In the first place comes the mode of life led by the child. One who is too carefully guarded from all possible dangers, who is kept in overheated and imperfectly ventilated rooms and allowed out of doors on only the finest days, soon acquires a hypersensitiveness of the mucous membrane of the nose and pharynx. In the same type of child profuse perspiration, owing to over-clothing by day and night, may be responsible for many colds. In such children the most trivial exposures to colds or draughts, or the wetting of the feet, will bring on an attack.

Another predisposing cause is undoubtedly adenoid vegetations in the nasopharynx.

Acute nasal catarrh is seen in *epidemics* during the cold months of the year: in measles, influenza, and diphtheria. It must be linked upon as due to a micro-organism acting possibly upon a mucous-membrane already the seat of some vascular disturbance.

Symptoms and Sequelæ.—The ordinary course of a "cold in the head" is too well known to necessitate a description. In infants it is usually attended by some rise of temperature, often with associated catarrhal inflammation of the digestive tract and bronchial tubes. The nasal obstruction may be so complete as to prevent the child taking the breast. Retropharyngeal abscess may develop. In older children coryza is often followed by bronchitis, sometimes by broncho-pneumonia. Sores on the upper lip and herpes labialis are common. Deafness or otitis media may develop by extension of the inflammation along the Eustachian tubes. The eyes may be infected by being contaminated with the nasal discharge. Many repeated attacks of coryza may result in chronic nasal catarrh.

The chronic runs a course of from three days to a fortnight. Apart from its possible complications it is not dangerous.

Treatment.—Prophylactic treatment lies in the avoidance of those errors in the mode of life which have been enumerated above. Continuously there arises the question of how to break the habit of catching cold already acquired. Should there be adenoids and enlarged tonsils, they should be removed, and little good will be done until this has been accomplished. If there is no local cause to be dealt with, it is difficult to change the child's habit of life until the summer-time comes. If in the warm weather a new regime be started, an out-of-door-life begun, and the various coddling measures withdrawn forbidden, by the winter the child will usually be able to withstand the additional risks that it will incur.

During an attack the child must be kept in a well-ventilated, warm room, and take only simple food. The upper lip and nostrils should be coated with vaseline at night, and a plentiful supply of dry bread-kneaders allowed. Hand blowing of the nose is not to be encouraged. The nostrils should be opened. Quinine, belladonna, or Dover's powder may be prescribed.

CHRONIC NASAL CATARRH.

This is a condition which is not to be neglected, as it may lead to permanent trouble in the ears, or to imperfect speech and respiration. It is due to a variety of causes. By far the most common and the first to be suspected is cases of chronic nasal discharge, is the presence of adenoid vegetations in the nasopharynx. Foreign bodies in the nose are not infrequent, and this possibility should be remembered, especially when the discharge is unilateral. Nasal polypi, and the various

deformities of the nasal passages, are rare causes. Repeated acute attacks may set up a chronic catarrh. Chronic nasal discharge occurring in infants is frequently due to inherited syphilitic disease (p. 178).

The *rhinarrhea* must be directed towards the removal of the cause of the symptoms. Without efficient local treatment, the nose syringing out of the nose with normal saline, and the administration of cod-liver oil or favourite foods, are of little avail.

CHRONIC RHINITIS.

Simple, hypertrophic, and atrophic rhinitis are so uncommon in children that the mere mention of their possible occurrence is sufficient. The two common forms are the mucous and syphilitic. These are described elsewhere: membranous rhinitis under diphtheria (p. 239), and syphilitic rhinitis with the other manifestations of inherited syphilis (p. 178).

II.—DISEASES OF THE TONSILS.

ACUTE TONSILLITIS.

Swelling and redness of the tonsils is a common condition seen at the outset of all the acute specific fevers and at least of the acute infections to which children are subject, such as rheumatism, enteric fever, influenza, pneumonia, erythema nodosum, and some other purpuric states. Occasionally it is seen early in acute polyomyelitis. In these infections it is associated with some general pharyngeal infection.

Occasionally it occurs with a less serious significance, being perhaps associated with nasal or bronchial or intestinal catarrh.

The symptoms and treatment are considered below under follicular tonsillitis.

The diagnostic points, such as they are, in the acute tonsillitis of the infections have been given in the descriptions of these diseases.

ACUTE FOLLICULAR TONSILLITIS.

Etiology.—This is the commonest form of acute tonsillitis. In its etiology the most important point is the undoubted connection between it and rheumatic manifestations. A definite acute attack of rheumatism may follow within two or three weeks of the tonsillar inflammation, or the sore throat may precede the muscular pains or swelling of joints by only a day or two. From this we must conclude that the damaged tonsil allows the entrance of the rheumatic organism; probably the tonsillitis is itself a rheumatic manifestation.

Apart from these rheumatic cases, follicular tonsillitis occurs at the onset of many infections, may arise from cold, or without any cause at all to which it can be attributed.

Symptomatology.—The symptoms are abrupt at their onset, often severe, and may precede any definite local signs. There is an initial feeling of chilliness, occasionally a rigor, often there are headache and general pains all over the body; vomiting, however, is not seen, except in infants. The temperature runs rapidly up to 104° , often to 104° or higher. The local signs are slight swelling of the tonsils, becoming more pronounced with general facial injection. Small isolated patches of exudate easily removed and re-accumulating rapidly appear on the tonsils to which they remain localized. They may coalesce and spread all over the tonsils. The glands at the angles of the jaw show only slight swelling and tenderness, often none at all. The disease is always bilateral. With a mild attack the only symptom complained of may be loss of appetite, which is in reality due to the pain caused by the swallowing of food.

The general symptoms are at their worst at the onset of the disease, and, rapidly lessening, disappear in three or four days. The local signs clear up a day or two later, but some enlargement of the tonsils often remains for a week or more. There is a great tendency to recurrences of the disease.

Diagnosis.—The rheumatic cases may be diagnosed by a history suggesting previous attacks of rheumatism; or by the presence of rheumatic manifestations.

The differential diagnosis from diphtheria has been given on page 238. From scarlatina and pneumonia the diagnosis is often in doubt for the first few hours. In older children vomiting is a strong point in favour of one of these two diseases being present.

Treatment.—In rheumatic cases salicylate of soda should be given, and with many this is the routine treatment for follicular tonsillitis. In the absence of any definite evidence of a rheumatic origin, a mixture containing iron and a saline aperient, or iron and potassium chloride, may be given. The tonsils should be opened at the outset of treatment by caloric. Headache or backache may be relieved by aspirin or phenacetin.

Locally, a gargle, spray, or douche of bichloride of mercury (1-5,000) or of potassium chlorate may be ordered. Potassium bichromate is of particular value in children, as they can swallow and suckling. One may be dissolved in the mouth every four hours. In infants, local treatment is best omitted.

In cases where there is a possibility of diphtheria or scarlatina, the child must be strictly isolated.

PERITONEILLAR ABSCESS (Quincy).

Quincy is not a common condition in children, and when it occurs resembles in every way the disease as seen in adults. It is only necessary to mention possible danger in these rare cases arising in infancy if spontaneous rupture is allowed to occur.

VINCENT'S ANGINA.*

Etiology.—This is a rare form of sore throat which is associated with the symbiosis of two organisms, a fusiform bacillus and a spirochete. The disease is only locally contagious. It is most common in patients between the ages of two and sixteen years, and is more frequent in the spring months than at other seasons. Oral sepsis is a doubtful predisposing cause.

Symptomatology.—The symptoms of the onset of the disease are in no way characteristic. They consist of sore throat, swelling of the glands at the neck, and headache. Vomiting is occasional; shivering is exceptional. A nasal discharge is common. During the later stages of the disease, although the local symptoms are severe, constitutional symptoms are absent.

The appearance of the throat changes as the disease runs its course. At first a membrane, which cannot be distinguished clinically from that due to the diphtheria bacillus, is present on the affected tonsil. The affection is usually unilateral, or more marked on one side than the other, but may spread to the soft palate or uvula. The corresponding lymphatic glands are enlarged, often considerably, but never suppurate. In the later stages there is much ulceration of the tonsil and a characteristic fetor of the breath is now present.

Diagnosis.—At first the disease can only be recognized by a bacteriological examination, so close is its resemblance to faucal diphtheria. Later, the severe ulceration, the fetor, and the absence of constitutional symptoms are characteristic.

Course and Prognosis.—The disease runs a prolonged course, an average one of about eighteen days. Healing is, however, complete, and death very rarely, if ever, occurs. Transient albuminuria and stomatitis are rare complications.

Treatment.—The affected area of the throat should be painted twice daily with saturated tincture of iodine. Should this fail, powdered methylene-blue may be applied to the ulcers. For the fetor, syringing with a solution of potassium chlorate and borax may be used. Internal medication is usually unnecessary.

* *Proc. J. B. Robinson, Brit. Jour. Child. Dis., July, 1920.*

CHRONIC TONSILLITIS.

A chronic hypertrophy of the tonsils is an exceedingly common morbid condition. In the great majority of cases enlarged tonsils arise from no definite cause that can be ascertained. They may be congenital and are often familial. As a rule they are associated with adenoid vegetations. Local causes do not often seem to account for the chronic hypertrophy. A tuberculous infection is exceptional. Although repeated attacks of acute inflammation are able to cause chronic enlargement, it is certainly true that more often the chronic inflammation predisposes towards the acute attacks.

The results and treatment of the condition may be considered in the following section on adenoids, with which the association is so close.

*III—DISEASES OF THE PHARYNX.***ADENOIDS.**

Etiology.—The causes of adenoids are wrapped in the same obscurity as those of chronic hypertrophic tonsillitis, and the many views which have been put forward cannot be said to have solved the problem of their origin. The condition may be present at birth, it runs in families, it is very rarely tuberculous, it has nothing to do with inherited syphilis and but little with rickets, it is more often the cause than the result of repeated colds in the head: these facts are undoubted, but beyond them we cannot go at present.

Symptomatology.—The symptoms resulting from chronic tonsillitis and adenoids, the common "T and A" of hospital practice, may be considered together. These may be taken under various headings.

1. **Catarrhal Symptoms.**—The "throat-cough" has already been described in detail (p. 321), and the tendency towards acute attacks of nasal and tonsillar disease has been mentioned. A chronic nasal discharge is very common; sometimes a chronic laryngitis is present. Conjunctivitis may be set up by direct and repeated infections of the eyes by the nasal discharge.

2. **Obstructive Symptoms.**—The voice may be of a nasal quality, and speech is often indistinct. Snoring at night is the rule, and breathing through the mouth is usual during the day and almost invariable at night. In the recumbent position the obstruction to respiration is increased, and thus restlessness at night and various nocturnal nervous disturbances (see below) are induced. More serious obstruction gives origin to deformities of the chest, especially in early life and in ricketty children. Of these the most common is the pigeon-chest, with its

prominent alveolar and straightened sides (p. 81). The apices of the lungs may show a poor percussion note and defective air-entry from imperfect expansion.

During infancy the obstruction to the breathing may interfere with suckling.

3. **Aural Symptoms.**—The most common of these is imperfect hearing, but actual deafness is very frequent. Pain in the ears and discharges from them are common, and arise from catarrhal or suppurative inflammatory changes in the middle ears.

4. **Nervous Symptoms.**—Affected children are often said to be abnormally stupid; certainly they frequently look so. The mental backwardness, when present, is probably due to the indistinctness of hearing or deafness from which they suffer, and to the various small illnesses and general poor health which tend to keep them from regular attendance at school. Such children, as a rule, can hear, but they do not hear distinctly—their answers, therefore, are slow, and this, with their indistinct speech and dull facial expression, makes them appear stupid.

In addition, there may be disturbances at night, when the obstruction to the breathing becomes more severe and the cough more troublesome. Restlessness has already been mentioned, and to the partial asphyxia occurring during sleep may certainly be attributed some cases of nightmare (night-terrors in neurotic children), nocturnal enuresis, asthma, and laryngeal spasm. The association with epileptic seizures is rather doubtful.

5. **General Symptoms.**—These are for the most part due to the obstructed respiration inducing an imperfect supply of oxygen to the blood, and to some extent to the repeated catarrhs of the nose and throat, disturbed sleep, and other symptoms from which these children suffer. At the same time it is probable that the persistent purulent discharge in the pharynx must produce some deteriorating effect, particularly when it is remembered how much of this is swallowed.

The general appearance of the patient is often highly characteristic. The nose is flat and broad, the nostrils narrow and often discharging and sore; the mouth open, and the front teeth protruding and overlapping. The facial expression is heavy and dull, and the complexion is pale. The body is badly held, the shoulders inclining forwards, the trunk stooping, and the whole appearance of the child one of listlessness and stupidity. Chronic enlargement of the glands of the neck is the rule.

Symptoms such as have been enumerated are rarely present during infancy; even in congenital cases they do not, as a rule, arise in any severity until the second or third years of life. The adenoids and enlarged tonsils frequently undergo atrophic changes about the time of puberty, and the worst of the symptoms may disappear then. The condition usually gives rise to more trouble during the winter than the summer months.

Diagnosis.—This ultimately rests upon the physical examination of the patient. On inspecting the throat, the tonsils are seen enlarged, red, and with an irregular surface. The enlargement may be of all degrees, up to that of such severity that the tonsils meet in the middle line. Adenoids may be suspected where there is chronic fossillar hypertrophy, and are certainly present when a thick crust of mucus is seen on the posterior pharyngeal wall. The question may be settled by digital palpation, when the soft masses can be felt. The finger, when withdrawn, is found covered with mucus and blood. This examination is easily carried out, but is not very often necessary for diagnosis purposes. In dealing both young children and babies, the examining finger should be smeared with glycerin. This simple device is often of great use, for when the finger is withdrawn the glycerin is tasted and the baby quieted. In older children there is a risk of getting the finger bitten. This is easily avoided by pressing the cheek in between the teeth when the mouth is open. The child will only attempt to bite as the finger is withdrawn, for with the finger in the nasopharynx the mouth is held widely opened. If the cheek is pressed in between the teeth, the child bites down upon its own flesh first, and so time is given for the withdrawal of the finger.

Treatment.—The indications for operation have here to be considered. Roughly speaking, they depend upon the symptoms produced by the presence of the abnormalities rather than upon the mere facts that the tonsils are enlarged and the nasopharynx contains adenoids. The most common indications are, then, persistent cough, repeated catarrhs, ear troubles, nervous disturbances, and chest deformities. As a rule it is only the slighter abnormalities that can be successfully treated by medical means.

Where the attempt is made to cure without operation, as where only slight symptoms, or none at all, are present, the throat and nose should be treated on the following lines: the enlarged tonsils may be sprayed or painted with astringent preparations, such as glycerin and iron or tannic acid. Iron and potassium chlorate may be given internally. The nose may be syringed through with alkaline lotions, and any discharge must be kept from accumulating within the nasal cavities. Most important is the encouragement of nasal breathing by means of exercises. The child is instructed to stand in front of an open window and take a series of deep inspirations through the nose, expiring through the mouth. This should be repeated two or three times in the day. The most successful line of treatment is to combine local treatment and tonifying courses with a change of air, preferably to the seaside. If no permanent benefit results, operation should be advised.

If the operations for enlarged tonsils and adenoids, the removal of them by the guillotine and curette is very successful if properly done. The recurrences are few after a thorough operation, especially if done after the child is three years old. About this age is the most

favourable for operation. The risks are few apart from those easily obtained by surgical cleanliness, the two unfortunate sequelæ which are most common are earache or ear discharge (nearly always only temporary), and broncho-pneumonia. The latter is, however, very infrequent.

RETROPHARYNGEAL ABSCESS.

This disease, although to be treated by surgical measures, is of great medical interest, because the diagnosis of it so frequently has to be made by the physician. The patient may come under observation for difficulty in breathing or in swallowing.

Etiology.—Retropharyngeal abscesses may be of two sorts—septic or tuberculous.

The septic class arises much more commonly in infants than in older children. It is caused by suppurative changes in the retropharyngeal glands, and thus may be due to infective nasal and pharyngeal conditions, acute nasal catarrh, adenoids, and the like. Sometimes the cause is not ascertainable. Occasionally scarlatina or measles may originate the condition. General malnutrition is a predisposing cause.

It is interesting to notice that the retropharyngeal group of glands is said to atrophy after infancy.

The tuberculous cases are of two kinds. The abscess may arise in connection with the breaking down of the retropharyngeal glands as before, the changes in them being tuberculous instead of suppurative. This type of case is again most frequent in infants, especially during the second year of life. In older children cervical spinal caries is the common origin of the condition.

Symptomatology.—Dealing first with the septic cases. The affected child is usually delicate and ill-nourished, but occasionally is of a robust type. There is more fever and loss of weight, and these may be present for several days before any local symptoms are seen. The early symptoms consist of difficulty in swallowing, a frothy salivation, nasal discharge and cough. Regurgitation of food through the nose may occur. The voice is nasal, hardly hoarse; articulation is indistinct. When the abscess arises at, or spreads to, a lower level, there is difficulty in breathing, usually continuous but occasionally paroxysmal. The mouth is generally held open and the breathing becomes noisy, especially during inspiration; later there may be loud inspiratory and expiratory stridor. Feeding increases the dyspnoea. There is a short expiratory grunting cry. Very commonly there is head-retraction, which may be quite marked. Torticollis is less often seen and probably depends on the laterally placed glands being inflamed. With the onset of respiratory obstruction there appear moist sounds in the lungs and inspiratory recession of the lower part of the thorax.

On digital examination of the pharynx a rounded, soft swelling may be felt, which is usually greater on one side of the middle line than on the other. It is often globular in outline, and can be felt to fluctuate. Occasionally, however, the abscess is situated too low down to be marked by the finger. On inspection the swelling may be seen if placed high up at the level of the base of the tongue, as is most common. There are injection and general swelling of the pharynx. In addition there may be swelling in the neck below the angle of the jaw and in front of the sternomastoid muscle.

In the tuberculous cases these symptoms and signs are also present, but the onset of the dysphagia is usually much more gradual, and where due to cervical caries, symptoms of the carious disease are as a rule well marked, and may have been present for a considerable time. In this group the collection of fluid is behind the prevertebral layer of deep cervical fascia.

Diagnosis.—The mistakes arising in cases of retropharyngeal abscess are due usually to the fact that the condition is not thought of, and so the throat is not digitally examined. It is impossible to over-emphasize the importance of examining the pharynx with the finger in any case of dysphagia or stridor, particularly in infants. In such conditions retropharyngeal abscess is the first disease to be suspected.

With a proper digital examination the diagnosis is usually easily made. The presence of a foreign body in the larynx or oesophagus, or laryngeal inflammation, catarrhal or membranous, are the chief alternative possibilities.

Prognosis.—Where undiagnosed, the abscess usually causes death. In wasted children it is a serious disease, and death occurs in from 5 to 10 per cent. of cases (Hall). It may be due to general infection or tetanus-præmonia, less commonly to anglyxæ. Retropharyngeal abscesses may recur, particularly where there has been a difficulty in draining efficiently the lowest parts of the abscess cavity.

Treatment.—The abscess must be opened as soon as its presence is diagnosed. In septic cases an incision should be made through the posterior wall of the pharynx, particular care being taken to drain the lowest part of the abscess. Tuberculous cases should be drained from without, through the neck.

IV.—DISEASES OF THE LARYNX.

CONGENITAL LARYNGEAL STRIDOR.

Etiology.—This is a condition which is much more common in girls than in boys. The symptoms arise very soon after birth, and persist

for about two years, when they pass off. No bad effect seems to be exerted upon the child's general health, and no particular association with rickets has been noted. Indeed, its appearance so early in life excludes this as a possible causative factor.

The cause of the stridor is not as yet fully apparent. It may be due to excessive involution of the epiglottis, so that the ary-epiglottic folds are brought close together, as in a case described by Dr. Lees. Other observers have ascribed the condition simply to excessive yielding of the laryngeal parts under the influence of hurried breathing, while others have held that the condition is entirely or in part spasmodic in origin. It seems probable that laryngeal spasm alone does not account for all cases, and it may well be that undue curving of the epiglottis, excessive yielding of the laryngeal structures, and spasm, may all be present.

Symptomatology.—As a rule there is perpetual slight inspiratory and expiratory stridor, together with inspiratory crowing; directly the breathing becomes more hurried or deeper than normal. In other cases, the breathing is quite natural until some extra deep breaths are taken, when the crowing sound is heard. The crowing, however, in either type of case is not associated with any respiratory distress or cyanosis, as it may be in laryngismus stridulus. The obstructive symptoms are usually absent during sleep, and are increased in any catarrhal condition of the respiratory tract.

Diagnosis is, as a rule, easily made from the fact that the condition dates from soon after birth.

Prognosis.—As has been mentioned, it does not affect the general development of the child, and the symptoms pass off, usually during the second year of life.

Treatment.—None is necessary apart from that directed towards the satisfactory growth of the child.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—Non-membranous laryngitis is not uncommonly seen in children, particularly in those under the age of five years. It may be caused by cold, and this is frequently associated with acute nasal catarrh, bronchitis, and broncho-pneumonia. It is one of the common catarrhal symptoms seen in the initial stage of measles, and may occur in whooping-cough, influenza, and scarlatina.

Symptomatology.—The symptoms may be mild, but are often of great severity. They consist of fever, a distressing hoarse cough, which is at its worst at night, and inspiratory stridor. Aphonia

is common. Where severe the laryngitis is associated with pallor of the face, cyanosis of the lips, retraction of the ribs, and partial collapse of the lungs. Broncho-pneumonia may develop. The course of the disease varies from three to fifteen days.

Diagnosis.—This is always difficult, owing to the possibility of the laryngitis being due to the diphtheria bacillus. Where there are remains of exudate on the fauces and markedly enlarged cervical glands, the diagnosis of diphtheria is almost certain. In catarrhal laryngitis the glands are rarely enlarged, and those in the neighbourhood of the cricoid cartilage should be carefully examined. Aphonia is unusual in catarrhal cases, common in diphtheritic. The presence of a blood-stained nasal discharge points towards diphtheria. Between catarrhal and primary laryngeal diphtheria the diagnosis is very difficult. The higher the temperature and the more sudden the onset of local symptoms, the more likely is the condition to be catarrhal; while the greater the symptoms of prostration, the greater the probability of diphtheria, although primary laryngeal diphtheria may be associated with very mild constitutional symptoms. In all cases cultures must be taken from the back of the throat (p. 249).

Koplik's spots will, as a rule, be found in those cases of laryngitis due to measles.

Prognosis.—This is grave particularly in infants and young healthy children, in whom severe collapse of the lungs tends to occur. Cases associated with measles and scarlatina are often of great severity. Frequently, however, rapid improvement occurs with treatment, and children who appear most seriously ill recover. The prognosis therefore be guarded, especially as there is the great difficulty of excluding diphtheria.

Treatment.—Even in slight cases the child should be carefully treated, and should be kept in bed. In severe cases the steam-kettle often gives prompt relief. It should not be put close enough to the child to cause fright, and is preferably kept at the foot of the bed. A tent may be used in order to keep away draughts, and by this means objections to an open window may usually be met. In this way the child breathes warm moist air which is fully oxygenated. Friar's balsam or creosote may be added to the water in the kettle. Light fomentations may be applied to the throat; mustard plasters should not be used, lest irritating fumes escape and be inhaled. The local application of leeches is of value. If the fever is high, ice compresses may be ordered instead of the fomentations.

The bowels should be opened by calomel. Small doses of antimony (5 drops of the wine) and ipecacuanha may be given. Where there is much prostration, an emetic may be of service. The right side of the heart may require relief and will probably need stimulants. If

the difficulty in breathing does not improve by this treatment, intubation or tracheotomy may become necessary.

Where diphtheritic laryngitis cannot be excluded, the child should be strictly isolated. A dose of antitoxin may be given, pending the result of a bacteriological examination.

SPASMODIC LARYNGITIS.

(*Laryngitis Stridulosa*.)

This disease is also known as catarrhal spasm of the larynx, or pseudo-croup. It is sometimes wrongly called laryngismus stridulus (p. 336). In it there is a slight catarrhal laryngitis, superimposed on which is laryngeal spasm.

Etiology.—The disease is seen in either strong or delicate children, between the ages of six months and three years. It is rare after five years of age. It is more common in boys than in girls. Predisposing causes are enlarged tonsils and adenoids, necks, and previous attacks. The attack usually comes on as the result of exposure to cold.

Symptomatology.—The child during the day may appear well, or may have a slight cold or be a little hoarse. Towards evening a cough develops, and at night the alarming symptoms appear. The child wakes suddenly, with difficulty at breathing and much distressed. There is loud stridor, especially noisy during inspiration. Hoarsity, hoarseness, and a metallic cough. The temperature, if raised at all, is not above 101°. The symptoms are aggravated by excitement. In three or four hours the condition becomes much improved, only slight hoarseness remaining. During the next day the child remains fairly well, but at night there is a reappearance of the severe dyspnoea. The third day is again passed in comfort, and in the night following the distress is much less, as a rule, than in the previous two. Some hoarseness remains for several days, but no further dyspnoic attacks occur.

In many cases the illness is not nearly so severe as the one described. The spasm at night is much less, and the distress produced is only slight and very transient.

Some children are prone to attacks of this disease throughout the cold season of the year.

Diagnosis.—When once the nature of the disease, that of slight laryngitis with added spasm, is appreciated, the diagnosis is easily made. It differs from acute catarrhal laryngitis in that the symptoms due to inflammation are only slight and are hardly noteworthy, and those due to spasm are added. From diphtheritic laryngitis it is differentiated in the same way. In neither simple nor diphtheritic laryngitis do we see the marked improvement during the day-time.

If there is any doubt as to the presence of laryngeal spasm, the point may be settled by noticing the effect of the inhalation of a little chloroform, which will allay the spasm and the symptoms due to it. In laryngismus stridulus there is no catarrh at all, no stridor, but actual cessation of breathing for the moment, followed by inspiratory crowing.

Prognosis.—While the disease is very alarming, it in reality is accompanied by no danger to life. As has been mentioned, one attack predisposes to others, until after the age of three or four years.

Treatment.—This should be on much the same lines as that of catarrhal laryngitis. The steam-bath is useful, cold air tending to increase the spasm of the larynx. Hot local applications and expectorants may be ordered, and should the spasm be very severe an anæsthetic may be given or a few inhalations of chloroform administered. Antipyrin is a useful drug for combating the spasm, and may be given in doses of a grain for each year of the child's age up to five grains; thus for a child of two years old two grains should be given. Chloral and bromide are of value.

During the day following the attack, the child should be kept in bed, the expectorant mixture given, and at night the sedative repeated.

After the disease has abated, hygienic and therapeutic measures should be ordered, with a view to bringing the child up to the best standard of health in order to prevent further attacks. Particular care should be taken to have any abnormal conditions of the throat removed, such as enlarged tonsils, adenoids, or an elongated uvula.

LARYNGISMUS STRIDULUS.

(*Laryngospasmus*.)

This is an entirely sporadic affection, and is described under the nervous diseases. For the sake of convenience, the chief points about it may be mentioned here, that no mistake may be made in its differentiation from catarrhal spasm of the larynx. It occurs in infants of about twelve months old, and is nearly always associated with rickets, and often with the other nervous signs of that disease,—namely, facial irritability, tetany, or convulsions. There is no catarrhal condition present in laryngismus stridulus, no hoarseness, no stridor. There is not so much a difficulty in breathing as a sudden cessation of respiration followed by a characteristic inspiratory crow, these attacks occurring suddenly, and being as a rule often repeated.

MEMBRANOUS LARYNGITIS.

Almost invariably this is due to the Klebs-Loëber bacillus, and has been described under diphtheria (p. 233). Membranous inflammation

of the larynx may be caused occasionally by other organisms, probably streptococci, but these cases generally follow injuries to the pharynx, such as scalds.

OEDEMA OF THE GLOTTIS.

Cases of simple oedema and inflammatory oedema may be considered together. The cases are numerous. The simple oedema is most commonly seen in association with renal disease. Inflammatory changes may be due to scalds, commonly caused by a child drinking out of a kettle or from a hot water tap. It may result from a snap-sting inside the mouth when eating fruit. Foreign bodies in the larynx may cause this condition, or it may arise in cases of retro-pharyngeal abscess, sleeping tonsillitis, erysipelas of the neck, and acute catarrhal laryngitis. Very rarely it is seen in connection with varicella or variola. The swelling occurs in the ary-epiglottic folds and in the epiglottis, and may completely occlude the air-passage.

The chief symptom is that of rapidly increasing dyspnoea, with the inspiratory difficulty more marked than the expiratory. The development of the dyspnoea is very rapid, and death from asphyxiation may occur in a few hours. The diagnosis is made by digital examination of the throat, when the large, round, swollen folds may be felt.

The treatment consists in scarification by the finger-nail, and the external application of ice. Tracheostomy must be performed if the symptoms do not abate. Where possible the cause should be treated.

CHRONIC LARYNGITIS.

Three chief forms exist—simple, tuberculous, and syphilitic. In addition, papillomata of the larynx may be associated with some chronic laryngitis.

Simple chronic laryngitis is always associated with adenoid vegetations, and is due to the chronic congestion of the larynx. The treatment required is the removal of the adenoids, after which the laryngitis quickly disappears. Apart from this no measures are of avail.

Tuberculous and syphilitic laryngitis are both uncommon. They are described on pages 128 and 129 respectively.

NEW GROWTHS OF THE LARYNX.

Papillomata form the great majority of the new growths of the larynx, and are not of great rarity. The symptoms may start during infancy, and consist of hoarseness, metallic cough, and stridor. Their development is very slow, and many months may pass before the stridor becomes at all distressing. The diagnosis is suggested by the fact that these symptoms are very gradual in their onset, and

that there are no indications pointing to chronic laryngitis. If it is confirmed by the examination of the larynx, a matter of course since the introduction of the direct laryngoscopic method. The prognosis must be guarded, because operation for the removal of the growth may be followed by laryngo-pneumonia, and more particularly because there is a great tendency to recurrence of these tumours.

The treatment consists in the removal of the growth.

FOREIGN BODIES IN THE LARYNX.

It is not an uncommon accident for a child to get a foreign body into the larynx, from which it may fall into the trachea or into one of the bronchi, usually the right. As a rule violent coughing is set up at the time, but often nothing of the sort occurs, and in these cases there is no suspicion in the mother's mind of what has happened. Or again, a small child may be left alone or in charge of a child half a little older than itself. All of a sudden there arise severe cough and dyspnoea, and no explanation is forthcoming. We have then three types of case: where the presence of a foreign body is known; where it is totally unsuspected; and where it may be suspected or at least cannot be excluded.

The results may be various. Remaining in the larynx or trachea, a foreign body may cause immediate death by asphyxiation or rapid death from acute oedema of the glottis, or may produce similar until it is removed or changes its position. When in the bronchus (usually the right), it may be associated with unilateral bronchitis, bronchopneumonia, collapse, bronchiectasis, abscess, or emphysema. After weeks or months it may be expelled by coughing.

Diagnosis.—Where the accident is known to have occurred, the diagnosis is already made, but where no initial symptoms have been produced, and the condition has been unsuspected, the diagnosis is one of extreme difficulty and is usually impossible. Where there is an acute onset of dyspnoea of unexplained origin, the possibility of the presence of a foreign body must be borne in mind. Direct laryngoscopy, possibly bronchoscopy, will be of use in some cases. Winterer says are rarely of use, as the foreign substances are not usually visible by them; nevertheless a trial of this method should never be omitted.

Changing and anomalous signs as the lungs should suggest this diagnosis. Shifting areas of collapse of lung or a unilateral bronchitis may be found. But little of help can be said apart from emphasizing the importance of remembering the possibility of a foreign body in any obscure chest case in a child.

Finally, it must be borne in mind that a large body, such as a coin or button, may give rise to laryngeal stridor, although not lodged in the larynx. This possibility must as far as possible be excluded by the passage of an œsophageal tube.

Treatment.—At the time of the accident the patient should be inverted to aid the expulsion of the foreign body by coughing. Tracheotomy may be necessary, and may save life where the obstruction to the breathing is in the larynx. If expulsion does not occur, various surgical measures will be needed according to the position of the foreign body, while it can be reached.

V—DISEASES OF THE LUNGS.

PULMONARY ATELECTASIS.

Collapse of the lung is very frequent in children. It may be considered under the two headings of congenital and acquired.

CONGENITAL ATELECTASIS.

In this condition some parts of the lungs, varying in extent, but usually including the posterior and basal portions, remain unexpanded after birth. It is usually seen in premature or delicate infants.

Two types of fatal cases may be described. In the first, the infant is born much asphyxiated, and the attempts to make it breathe well and cry loudly are only partially successful. The child remains cyanosed, and dies after a few days. In very weakly children the cyanosis at birth may be only very slight. In the second type, the initial asphyxia is as a rule severe, but the resuscitating efforts appear successful. The child, however, remains very delicate, does not thrive, and is liable to attacks of cyanosis with a subnormal temperature. These come on suddenly, and any one of them may prove fatal even after several months of life. Usually no cause can be found to which the cyanotic attacks can be attributed, but sometimes they are due to overdistention of the stomach.

On the other hand, many cases do not end fatally. A delicate infant liable to cyanotic attacks may get quite well, and the tendency is for them to disappear when some gain of weight has occurred.

Diagnosis.—This depends rather upon the symptoms than upon physical signs. There are, however, as a rule, areas of impaired resonance and defective aërcuity, and possibly a few crepitations at the bases of the lungs.

Treatment.—Full expansion of the lungs at birth must be encouraged by rubbing, bathing, or supporting the infant until its cry is loud. In the general treatment of these delicate babies the two chief points are the maintenance of the body-heat by hot-water bottles or an incubator (70°–80° F.) and careful dieting. It is, however, of great importance that these children should be taken out of bed once or

twice a day, and made to cry well in order to expand the lungs. The worst sign in this is the continuance of the feeble cry.

During a cyanotic attack, a mustard bath (Appendix A) should be given and the surface of the body stimulated by friction. Artificial respiration should be employed when necessary. Oxygen and brandy are valuable.

ACQUIRED ATELECTASIS.

Etiology.—This is a very common condition in children, and often tends to cause aggravation of the symptoms of the illness in which it occurs. It is predisposed to by general enfeeblement, rickets, and deformities of the chest.

Obstruction to respiration is the common cause for collapse of lung tissue. Favourite sites for collapse are the basal portions of the lungs posteriorly and the middle lobe of the right lung. Not uncommonly a fringe of collapse about an inch wide is seen round the edges of the bases of the lung, but this is of little importance.

Obstruction to respiration may be caused by disease of, or in close connection with, the respiratory organs, or by morbid conditions entirely apart from these. Under the first heading will come the common causes, bronchitis, bronchopneumonia, laryngitis, retro-pharyngeal abscess, foreign bodies, enlarged bronchial glands, and others which will suggest themselves to the mind. Of the diseases other than respiratory giving rise to pulmonary collapse, the most important are general enfeeblement, and abdominal distension as from ascites, acute dilatation of the stomach, or paralytic distension of the intestines. The yielding chest-wall of rickets and various deformities of the chest will predispose to collapse, as has been mentioned above.

Collapse of the lung may be due to compression, of which the most frequent instance is the collapse caused by pleural effusions. Some cases of this variety need emphasis. In children, enlargement of the heart, when at all marked, is nearly always associated with compression of the lower lobe of the left lung, and mistaken diagnosis of pleural effusion, pneumonia, or infarction may be avoided if this fact is remembered. The same occurs in pericardial effusion. In a lobar pneumonia causing consolidation of the upper lobe of the lung, the lower lobe may be compressed by the weight of the affected lobe. In an interlobar empyema very purulent and cholesterolic signs may be found, due to collapse of various portions of the lung.

Lastly, must be mentioned cases of collapse of lung which may occur in children without anything being found to account for them. In such, a lobe, or even the whole of one lung, may be involved.

Symptomatology.—These are dyspnoea and cough, with possible cyanosis. As a rule these occur as an aggravation of the symptoms already present from the associated disease.

The physical signs vary according to the extent of lung involved. Very small or superficial areas of collapse are not recognizable. Over rather larger areas, the percussion note is impaired, the air-entry and vocal resonances are diminished. Distant bronchial breathing and crepitations may be heard. Where a whole lobe or more is collapsed the signs simulate those of pleural effusion. There are dullness and much diminished or absent air-entry. The affected side of the chest, however, is smaller than normal.

Diagnosis.—Where atelectasis may be suspected the diagnosis is as a rule easily made, and thus the great frequency of pulmonary collapse in children must be remembered. In a case of severe bronchitis it may be very difficult, however, to decide whether the signs of consolidated lung arise from collapse or broncho-pneumonia. Often the diagnosis cannot be made without watching the course of the disease, for the signs of early pneumonic consolidation are exactly simulated by collapse. The persistence of poor air-entry and the absence of bronchial breathing will be in favour of collapse, while the sudden disappearance of all signs of consolidation after a fit of crying or coughing will indicate that it has been present. In collapse of large extent, the physical signs suggest a diagnosis of pleural effusion, but there will be a diminution rather than an enlargement of the affected side of the chest, the interspaces will not be protruded, and there will be no dislocation of the heart away from the dull side of the chest.

Prognosis.—The outlook in various respiratory diseases may be rendered worse by pulmonary collapse. The prognosis in acquired atelectasis is that of the associated disease. Of itself, collapsed lung tends to get well, either rapidly or slowly. The chief bar to complete recovery is the presence of pleural adhesions. Uninfected portions of lung tissue are probably predisposed to tuberculous infection.

Treatment.—For the most part this consists of treating the cause of the collapse, and in rickety or delicate children any respiratory disease must be very strictly treated in order to prevent the onset of collapse so far as possible. It is to be remembered that a certain amount of crying is good for children in whom collapse may be threatened. By examining children who are very feeble, or who have bronchitis or broncho-pneumonia, crying is frequently induced, and it is often a mistake to try to check it.

ACUTE BRONCHITIS.

This is a common disease in children. Owing to the frequency with which it leads to the more serious conditions of broncho-pneumonia and pulmonary collapse, it always needs careful treatment.

Etiology.—Bronchitis occurs in many infections, notably measles, whooping-cough, influenza, and typhoid fever. It is commonly present in cases of acute nephritis.

The organisms causing bronchitis are not as yet known. From a consideration of post-bronchitic pneumonia and its complications, it is probable that streptococci and pneumococci are the usual causes of bronchitis. That the unrecognized organisms of measles and whooping-cough are directly responsible for the bronchitis in those diseases is a view widely held, but it is more probable that streptococcal and pneumococcal infections, particularly the former, produce the condition in these diseases. In a certain number of cases clinically resembling bronchitis, tubercle bacilli are present in the sputum when this has been obtained by passing a swab into the pharynx (Koplik).

The predisposing causes of bronchitis are numerous. Exposure to cold is important. In many cases, however, it is the "coddling" processes in which the child is subjected which are most to blame. Hot rooms, heavy clothing, lack of fresh air and exercise, aiming at the avoidance of all possibilities of chill, render the child susceptible to the most trivial exposure. On the other hand, the vagaries of the "hardening" parent are often responsible for acute bronchitis in children. In these cases it frequently follows a cold in the head.

In infants, sickness is one of the most common predisposing causes of bronchitis, and is often the underlying reason for repeated attacks. Pandal distention and diarrhoea are better regarded as due to ectasias associated with bronchitis, than as causes of it.

Enlarged tonsils and adenoids predispose to bronchitis by harbouring organisms in the nasopharynx and probably by the prevention of nasal breathing.

Attacks of acute bronchitis are common in certain chronic pulmonary diseases. Of these may be mentioned bronchiectasis, pulmonary tuberculosis, chronic bronchitis, and emphysema.

Symptomatology.—The symptoms depend upon the depth to which the inflammatory processes extend within the lungs. When the larger tubes only are involved, the symptoms are slight, while the smaller tubes are inflamed, the severity of the disease is increased.

In many cases the trachea and larger bronchi alone are affected, usually secondary to nasal or pharyngeal inflammation. Some cough is present, but there is little or nothing in the way of constitutional symptoms, and beyond a few moist crackles in the chest there are no physical signs of disease. Older children may complain of a sensation of rumour under the mandibular on coughing.

In most cases, however, the medium-sized and smaller tubes are involved, and there are fever, cough, and dyspnoea.

The symptoms of bronchitis develop rapidly even when preceded for a day or two by a slight cough. The temperature runs up to 100° or 101°, and in young children may go to 102° or 103°. High fever is

not uncommon in young children with bronchitis, and its presence does not always indicate pneumonia. With the fever the child is fidgety and restless. The skin is usually moist and is prone to show blotches and sweat rashes. A dry pleurisy may be present even in the absence of pneumonia.

The cough is at first dry and hacking. In the later stages it becomes loose and sometimes spasmodic. During convalescence it becomes more and more confined to the time immediately following sleep. There is seldom any expectoration, as all phlegm is swallowed, in the later stages, when loose and purulent, some may be coughed up or brought up with vomiting.

Dyspnea is seen when the smaller tubes are affected. The respiration-rate is quickened, the ribs rise and sink, and a short expiratory grunt with inverted respiratory rhythm is present in severe cases. In the worst instances cyanosis, orthopnea, and pulmonary oedema develop.

In infants and young children catarrhs of the alimentary tract, stomatitis and gastro-enteritis, are often associated with the attack of bronchitis. In any there may be nasal, pharyngeal, or laryngeal catarrh. Conjunctivitis is present in a considerable number of cases.

The physical signs in the chest need not be detailed, as they are similar to those seen in adults with the same disease. Acute emphysema, pulmonary collapse, and broncho-pneumonia are more prone to develop in children than in older subjects.

Capillary bronchitis or inflammation of the smallest tubes throughout the lungs, hardly warrants a separate description, as closely it is allied to broncho-pneumonia. Indeed, unless death supervenes at a very early date, pneumonia is always present in cases of capillary bronchitis. The symptoms are those of urgent dyspnoea, cyanosis, and collapse, and the physical signs are those of fine-tube bronchitis, in which deficiency of aeration all save the lungs is the most marked feature.

Diagnosis.—The differential diagnosis between bronchitis and post-bronchitic pneumonia, a matter which may be of considerable difficulty, is discussed on page 325. The ashy-grey pallor, the course of the disease, and the possible enlargement of the spleen usually serve to distinguish acute milary tuberculosis of the lungs from a simple acute bronchitis.

Course and Prognosis.—In favourable cases the fever disappears within three or four days, but it may be protracted for a week. It is irregular in type, and the nocturnal rises of temperature gradually become less and less marked as the patient's condition improves.

The chief dangers of bronchitis are those of pulmonary collapse and broncho-pneumonia. These are especially liable to develop in infants and young children. Rickets, by producing a soft and yielding

chest-wall, predisposes strongly to these complications. Capillary bronchitis, as already stated, is a very fatal condition, which, if it fail of itself to cause death, is invariably associated with pneumonia.

Treatment.—The treatment of lobaritis may be most conveniently considered with that of consecutive broncho-pneumonia (p. 361).

CONSECUTIVE (SECONDARY) BRONCHO-PNEUMONIA.

Introductory.—Primary pneumonia, which is in the great majority of cases due to the pneumococcus, has been described in the section devoted to the consideration of the pneumococcal infection (p. 321). As has been pointed out, it is a disease which, although the pulmonary lesions may be lobar or lobular in type, arises without any antecedent illness. The lung is infected through the blood-stream as most if not in all cases, and the pneumonia is in no way the result of an extension of inflammatory changes from the upper respiratory passages into the alveoli of the lungs. With it there is little or no generalized bronchitis. In its clinical course the disease shows certain characteristics. Its onset is sudden, its symptoms are due to the toxæmia, rather than to the extent of the pulmonary lesions, its course is self-limited, recovery is rapid by crisis or lysis, and as in other pneumococcal infections, notably of the pleura, pericardium, or meninges, no scar is left.

A consecutive or secondary broncho-pneumonia shows differences from a primary pneumonia in nearly every point which has been mentioned. It is always of the broncho-pneumonic type, and is never a truly lobar pneumonia. It arises as the result of the spread of inflammation from the upper parts of the respiratory tract into the pulmonary alveoli. As such it is nearly always associated with and consecutive or secondary to a recognizable bronchitis. The lung is affected through the respiratory tract, and not through the blood-stream. Its onset, although rapid, is never sudden, and it does not terminate by crisis. Its symptoms are those of pulmonary inadequacy rather than of severe toxæmia, and bacterial complications are not so commonly present in this disease as in primary pneumonia.

Such differences as these preclude as in attempting to separate as rigidly as possible the two great classes of pneumonia, the primary and the consecutive. Bearing in mind that many cases of primary pneumonia are now known to be broncho-pneumonic in distribution, it is seen that the classification of pneumonias in children into lobar pneumonia and broncho-pneumonia is no longer of any clinical value (p. 321).

The term secondary broncho-pneumonia is generally used to denote such cases as are secondary to other diseases such as, bronchitis, measles, whooping-cough, diphtheria, diphtheria, and marasmus. The objections to this use of the term are, firstly, that all cases arising in

connection with these conditions would be classed together, even though the pneumonia itself contained to the primary type; and secondly, that in this sense it does not sufficiently emphasize the fact that the pneumonias classed under this heading are all the result of extension from disease in the upper respiratory passages. If by "secondary" were meant secondary to disease elsewhere in the respiratory tract, the antithesis between primary and secondary cases would be complete, and our nomenclature in this matter at least satisfactory. Until such time come, it seems better to make use of the term "consecutive," as bearing the meaning of "consecutive to disease elsewhere in the respiratory tract." This group has also been described as "post-bronchitic" pneumonia, but such a term has the disadvantage that in some cases (e.g., the diphtheritic), bronchitis is not clinically recognizable.

These terms—secondary, consecutive, and post-bronchitic—have all been suggested as synonyms by Dr. S. West, who first described the primary form of lobar pneumonia which occurs in young subjects.

Etiology.—Consecutive broncho-pneumonia then is the result of the extension of inflammation from the respiratory passages to the pulmonary alveoli, and as such is seen in cases of simple bronchitis and associated with whooping-cough, measles, influenza, diphtheria, and occasionally during the catarrhal stage of posterior lobar meningitis and other infections. Septic broncho-pneumonia may arise from retropharyngeal abscess, the presence of a foreign body in the upper respiratory passages or œsophagus, septic tonsillitis and other conditions.

There are many causes which predispose towards this form of broncho-pneumonia. Rickets is of great importance in this respect, owing to the bronchitis, wasting, and softening of the chest-wall which are attributable to this disease. Wasting itself predisposes strongly to it, and a consecutive broncho-pneumonia occurs as a terminal event in a great number of cases of severe wasting. The malnutrition may be the result of simple marasmus, inherited syphilis, tuberculosis and the various septic infections of infants. Cold weather, over-crowding, and poverty, will predispose towards this form of broncho-pneumonia by tending to produce bronchitis, rickets, and malnutrition.

The *aggravation* of 84 cases, excluding those due to whooping-cough, measles, and diphtheria, is shown in the accompanying figure. The cases were drawn from the records of the Paddington Green Children's Hospital. The largest number occurred during the second year of life, as in the case of primary pneumonia (p. 281). In consecutive broncho-pneumonia, however, the incidence during the first year is seen to be very high, while no instances occurred in the records examined after the end of the first half of childhood.

The pneumococcus is present in rather more than half the cases, but usually in association with a streptococcus. In many cases, notably in

THE DISEASES OF THE RESPIRATORY SYSTEM

these complicating troubles and rhinorrhoea; the only organisms present is a streptococcus. Many other bacteria may produce the condition. The Klebs-Löffler bacillus is responsible for the most fatal form of the disease.

Symptomatology.—As has been mentioned, the symptoms of consecutive pneumonia are those of a pyrexemic disease rather than of a pure toxemia, as is often the case early in primary pneumonia. They consist chiefly of increased bronchitis increased in their severity.

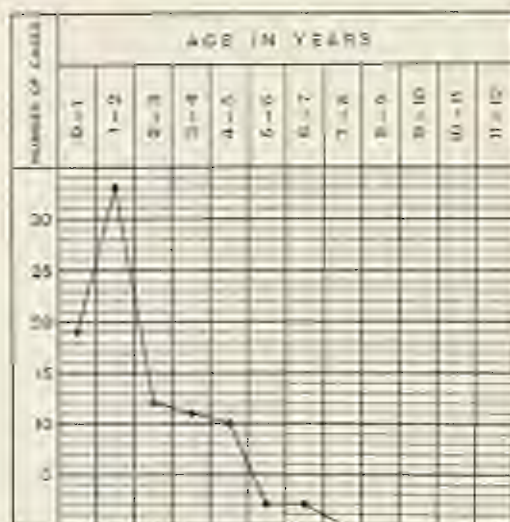


FIG. 1. *M. pneumoniae* is associated with pneumonia in 100% of cases. (From the report of the U. S. Public Health Service, 1917.)

Toxic symptoms are of course present, although playing a rather part in the symptomatology of the disease.

The onset of the disease is rapid, but is never as sudden as in the case of a primary pneumonia. It usually occurs during the first week of bronchitis, and is marked by an aggravation of the existing symptoms.

The temperature is neither so high nor so sustained as in primary pneumonia. As a rule the chart shows a difference of three or more degrees between the morning and evening temperatures. With the fall of temperature in the early morning there is often much sweating, and the dry, burning skin of a pneumococcal pneumonia is absent.

The cough is never absent, as it may be early in the primary form.

and throughout the illness is a severe symptom. At first it is dry and hacking, but later becomes looser. Even then, however, it is severe and often spasmodic in character, and may cause vomiting.

Respiratory distress is a marked feature of the disease. Actual dyspnea is present rather than the tachypnea (heightened respiration-rate without respiratory distress) of primary pneumonia. The dyspnea depends to a large extent upon the amount of accompanying bronchitis, and is often extremely severe. The respiratory rhythm is reversed, the pause occurring while the lungs are expanded, and a short expiratory grunt is frequently present. The ribs may dilate during inspiration, and in bad cases there are respiratory movements of the lower lip, jaw, and head. Cyanosis is frequently present, and other signs of right heart distress and failure may be added. Death occurs with edema of the lungs; pallor, sweating, drowsiness and groaningness.

The nervous symptoms which may be so conspicuous in primary pneumonia are far less common in consecutive broncho-pneumonia, and when they occur they are as a rule seen towards the termination of a fatal case. Vertical suppurative meningitis may develop.

The catarrhal symptoms which have been noted as occurring in acute bronchitis are often still more conspicuous in consecutive broncho-pneumonia. Of these the most important is diarrhea, which is very frequently present. It may be of a severe type, and may seriously add to the gravity of the child's condition. It is particularly prone to develop in eclctic infants with this disease. Nasal catarrh, stomatitis, pharyngitis, and laryngitis may also be present.

In severely wasted infants the symptoms pointing to broncho-pneumonia are very commonly extremely ill-marked, and often those of the condition passing unrecognized during life.

Physical Signs.—The bases of the lungs are the constant situations of the areas of consolidation in consecutive broncho-pneumonia. It is rare to find the apices attacked unless consolidation is present elsewhere in the lungs. More than one area of solidified lung is commonly present.

Bronchitic signs throughout the lungs are recognizable, although some diphtheritic and necrotic cases are exceptional in this respect. Acute emphysema is usually present, especially on the anterior aspect of the chest. The aspect of the chest is often that of forced respiration, owing to the temporary condition of emphysema.

The signs over the pneumonic areas are commonly distinctive of consolidation, consisting of dullness, bronchial breathing, bronchophony, and consonant rales. In other cases, to which some would limit the term *catarrhal pneumonia*, the pneumonic area is only recognizable by the metallic and loud character of the crepitations, the other signs of consolidation not being developed. A pleural friction is occasionally heard.

Clinical Varieties.—Diphtheritic and industrial cases have been described on pages 242 and 248 respectively. The pneumonia of measles and whooping-cough is in most cases of the consecutive type, primary pneumonia being here exceptional. The course of the consecutive broncho-pneumonia is often very protracted in these infections. The characteristic cough of pertussis may disappear during the pneumonia, to reappear during convalescence.

Prolonged cases of consecutive broncho-pneumonia are very common, and add considerably to the danger of the disease. Should the croup, bronchiectases and fibrosis of the lung develop in a few instances.

In the cases associated with severe wasting or with inherited syphilis, the symptoms pointing to the pulmonary disease are rarely well-defined, as has been already mentioned. Broncho-pneumonia may be said to constitute the natural method of termination in a fatal case of chronic wasting.

The septic type of disease, the courses of which have been given, is also almost necessarily fatal. Its diagnosis during life is often a matter of considerable difficulty.

Complications and Sequelæ.—The complications of primary pneumonia, such as erysipela, pericarditis, meningitis, and otitis media, also occur in consecutive pneumonia, but in a considerably lower proportion of cases. Abscess and gangrene of the lung may occur. Diarrhoea is to be classed as a symptom rather than a complication of this disease.

Of the sequelæ bronchiectases is that most frequently seen, and should it develop, is usually but not invariably persistent.

Diagnosis.—**From Primary Pneumonia.**—The differences from this shown by consecutive broncho-pneumonia have been fully described in the introductory section (p. 344).

From Bronchitis.—To say when an attack of acute bronchitis has developed into one of consecutive broncho-pneumonia is often at first a matter of great difficulty. The symptoms are the same in both conditions, and it is rather by a decided increase in their severity that the presence of pneumonia is recognized. A further increase in the fever and dyspnoea, and a further disarrangement of the pulse, respiration ratio, point towards the disease having reached the pulmonary stage.

The physical signs in the chest are usually distinctive, but they may be absent or simulated by areas of collapse. Signs of consolidation, however, where well-marked in a case of bronchitis, are more commonly seen in broncho-pneumonia than pulmonary collapse. The various signs which may be given by broncho-pneumonia have already been described.

The safest guide to a correct diagnosis in cases of doubt is the course of the disease. Where coincident manifestation of the symptoms

has failed to occur within four or five days, broncho-pneumonia is almost certainly present.

From Acute Military Tuberculosis of the Lungs. A simple broncho-pneumonia is usually distinguishable with fair ease. Severe pallor, intense ulcers, and prostration, signs of tuberculosis elsewhere, all point towards the former. In infants, if the lung condition is due to acute tuberculosis, the meninges will almost certainly be rapidly involved.

From Tuberculous Broncho-pneumonia, the disease is distinguished with much more difficulty, and often the correct diagnosis is only made post mortem. Spreading of the pulmonary process, with signs of calcation elsewhere in the lungs, and evidences of tuberculosis in the other organs, are the most important guiding-points. The course of the disease, the wasting and general appearance of the child, may be very similar in both conditions. In cases of doubt an effort should be made to collect the sputum from the back of the mouth by means of a sterile syringe on a holder, and a bacteriological examination for tubercle bacilli should be made.

Course and Prognosis.—In consecutive broncho-pneumonia the course is much less regular than in the self-limited primary disease. Lasting often two or three weeks, and exceptionally as many months, the disease comes to an end gradually, the symptoms slowly abating and the physical signs clearing up later.

The disease is a very fatal one amongst the children of the poor, particularly in infancy. In addition to the age, the general condition of the patient is of importance as a guide in prognosis, both nursing and sputum making the outlook much worse. Diarrhoea is often a serious symptom, and frequently turns the scale against the patient. Protracted attacks are more fatal than those coming to a rapid termination. The septic, diphtheritic, and malarial cases are the most fatal varieties, the first two necessarily causing death.

Death in consecutive broncho-pneumonia is in most cases due to right heart failure or to emaciation or diarrhoea. The suppurative complications so commonly causing death in primary cases account for a much smaller proportion of fatal results in consecutive broncho-pneumonia.

Treatment of Acute Bronchitis and Consecutive Broncho-pneumonia. When the temperature is raised, the child should be put to bed. The room should be warmed by an open fire, but all windows must be prevented and free ventilation ensured. For this reason the use of bed-curtains is not to be encouraged. The windows of the room should be kept open—at all events at the top—night and day, except when the patient is uncovered for examination or the changing of his clothes. As few people as possible should be allowed in the room at a time. A pocket of Gargol tissue is of use in cold

weather, and may reconcile the parents to the open windows. The child's legs must be kept covered, preferably by woollen garments.

A steam-kettle to warm and moisten the air is of use when there is laryngitis, or when in the earliest stage of bronchitis the cough is quite dry and irritable. It is of particular value when the atmosphere is cold and dry, as during the prevalence of easterly winds. As soon as the bronchial secretion becomes loose its use should be discontinued. The kettle should always be kept well away from the child, and is most conveniently placed at the foot of the bed.

The diet must consist mainly of milk. To this for children of a suitable age some yolk of egg or lighter mucinaceous foods may be added. As much milk as can be digested should be given, but in these cases the tendency to diarrhoea must be borne in mind. The presence of that symptom may necessitate a change in the diet given, even to the complete withdrawal of milk for a time.

At the outset of an attack of bronchitis the bowels should be opened by a mild purge. Remembering the danger of diarrhoea, a violent purge should not be used. A small dose of castor oil is perhaps the best, and should be given even in the presence of diarrhoea. Colours in small repeated doses may be substituted for it if necessary.

In the early stages, where the cough is dry and irritating, and there is little bronchial secretion, counter-irritation to the chest is useful. This may be given in the form of light linseed poultices, which certainly seem comforting, and are especially useful where pleurisy is present. In slighter attacks the chest may be rubbed night and morning with a liniment consisting of equal parts of the pharmacopœial lin. terribilina and olive oil. During the same stages expectorants should be ordered with a view to stimulating the bronchial secretion. A useful mixture is as follows:—

R. Vin. Ipecac.	℞ij	Syrup. Tolu.	℞x
Liq. Ammon. Acet.	℞ss.	Aq. Decid.	℥i 3j

This may be given every four hours to a child of one year. Where the cough is particularly dry, potassium iodide in $\frac{1}{4}$ -grain doses may be usually added to the mixture. Antimony is a valuable drug with more. Two or three drops of the wine may be used, replacing the ipecacuanha in the above mixture. It need not, of course, be given only in the earliest stages of the disease.

As soon as the bronchial secretion becomes loose, the steam-kettle should if have been used, and the poultices are best discontinued, the Dampree-tissue patch being retained. For drugs we must now rely chiefly upon ammonium carbonate, $\frac{1}{2}$ -grain doses of which may be added to the ipecacuanha mixture already given. Potassium iodide and antimony are now contraindicated, and the liquor ammon. acetis. is even better omitted. Squill is of value.

Should the bronchial secretion become too free and, by blocking the smaller tubes, lead to an increase of the dyspnoic symptoms, we

may endeavour to get rid of it by the use of the respiratory stimulant ammonium carbonate in larger doses, or check its production by belladonna. The latter is a valuable measure, particularly where the power of the cough is becoming feeble, but it is necessary to use it in large doses; as a rule flushing of the skin and dryness of the mouth should be induced by it for its most beneficial action to be obtained. Two minims of the tincture may be given to an infant of one year, and repeated every three or four hours until the desired effect is produced. Emetics may be of value at this stage, but often they fail to act. Repeated one-drachm doses of ipecacuanha wine may be ordered, but it is wiser to rely only on such mechanical means as tickling the fauces with a feather or producing retching by means of a spoonful. Medicinal emetics may increase or set up diarrhea.

The fever in bronchitis and consecutive broncho-pneumonia rarely needs treatment. Where necessary, sponging may be adopted in order to reduce the temperature.

The heart will require treatment in many cases of bronchitis, and in most instances of consecutive broncho-pneumonia. In the latter hardly is usually best given throughout the disease. For a child of one year, up to one ounce may be given in the twenty-four hours. Strychnine, digitalin, and atropine may also be needed. The relief of the right heart by means of leeches may be of the greatest value. Oxygen may be required and in bad cases should always be used. The indications for and methods of using the cardiac measures have been discussed under primary pneumonia (p. 30).

In cases of severe collapse, nothing is of more value than a hot mustard bath (Appendix A).

Diarrhea must be prevented so far as is possible by careful dieting and the avoidance of purgative drugs. Should it arise, a dose of castor oil may be given, and may be followed by a mixture containing 7 to 3 minims of the same drug. In protracted cases bismuth may be given, and should the state of the lungs and heart allow of it, opium may be ordered in small doses. Where the diarrhea is severe, milk may have to be omitted, and albumen water and bread substituted for a day or so.

During Convalescence a change of air is most beneficial. Tonics should be given. In particular, iron, if present, should be carefully treated.

CHRONIC BRONCHITIS.

In children chronic bronchitis is uncommon. In rocky children, acute attacks may lead to a chronic condition. More frequently chronic bronchitis is associated with pulmonary tuberculosis, interstitial pneumonia, bronchiectases and with heart disease. Occasionally it is seen in association with the emphysema resulting from asthma.

Symptomatology.—Constant cough, at its worst is the only distressing or the chief symptom. There is only slight expectoration. Other symptoms may be added from the disease associated with the chronic bronchitis. A tendency to catch "colds on the chest" is usually marked, and very severe attacks of acute bronchitis may arise in these chronic cases. The symptoms are usually at their worst during the winter months. Attacks of febrile and asthmatic nature are frequent.

Diagnosis.—It is often a matter of difficulty to exclude pulmonary tuberculosis. Anæmia, persistent loss of weight, nocturnal rise of temperature, and variations in the sputum index are in favour of tuberculosis. The sputum must be examined for bacilli.

Prognosis.—In young children with rickets complete recovery occurs as they grow up. In others the prognosis depends upon the disease underlying the condition.

Treatment.—The primary disease must be treated and the general condition of the patient improved as far as possible. A warm, equable climate is the most suitable for these children, and often is the only thing that benefits them. An emulsion of cod-liver oil with a few drops of *Ipecacuanha* wine may be given. Inhalations of eucalypti are useful. Alkalies may be given in the evening to ease the cough, and at night small doses of heroin may be ordered if necessary. Main reliance must, however, be placed upon the general measures. Enlarged tonsils and adenoids should be removed.

FIBRINOUS BRONCHITIS.

This rare condition is occasionally seen in children, and for a description of it the reader is referred to text-books of medicine dealing with diseases of the lungs. It is mentioned here only to emphasize two points concerning its diagnosis. In cases of diphtheria fibrinous casts of the bronchi are occasionally expectorated with vomiting. In children in whom epistaxis has occurred during sleep, blood clot, divided and branched, may later be expelled.

BRONCHIECTASIS.

(*Intestinal Permeosis*; *Pulmonary Fibrosis*.)

These may be considered together, for in children they invariably co-exist.

Etiology.—Most commonly bronchiectasis dates from an attack of *permeosis*, either primary or consecutive in type. The greatest danger of bronchiectasis arises in those cases of *permeosis* which run

a protracted course, or are associated with whooping-cough. In some cases it arises in connexion with chronic pulmonary tuberculosis, but it is to be remembered that this infection plays but a small part in the production or course of bronchiectasis in children. The presence of a foreign body in a bronchus is an infrequent cause of the condition. A few cases are said to be congenital, but these are certainly very rare in clinical medicine. Fibrosis may follow congenital anelastosis.

The condition described by Dr. Shukry as *acute bronchiectasis* is an uncommonly found in autopsies upon young children who have died of broncho-pneumonia and whooping-cough.

In its advanced stages bronchiectasis is seen chiefly in older children.

Symptomatology.—In early cases the signs of the previous attack of pneumonia remain, the rales becoming loud and resonant, while the sputum is increased in amount, purulent, and sometimes bloody. In the acute bronchiectasis of young children no sputum is expectorated, and the condition can hardly be more than surmised by the non-resolution of the lung and the loud character of the breath-sounds and rales.

In the advanced cases of older children the signs are those seen in adults. There are falling-in of the chest, cyanosis, marked clubbing of the fingers, dislocation of the heart towards the affected side, together with the signs of dullness and cavernous breathing usually at the base of the lung. Where the process starts in the upper lobe, as in such cases as follow an apical pneumonia, the lower parts of the lung usually become involved later. In the well-marked cases the breath is offensive and the sputum highly fetid.

There is in bronchiectasis a great tendency towards repeated attacks of acute bronchitis, which may be serious in causing failure on the part of the chronically embarrassed right heart.

Morbid Anatomy.—The only point to be mentioned here is one which has a bearing upon the surgical treatment of the disease in children. In young subjects the dilatation of the bronchial tubes is almost invariably uniform, and involving many divisions of the bronchus. A sacculated dilatation is at this age extremely rare, and a single large dilatation is not found.

Diagnosis.—The exclusion of pulmonary tuberculosis is the most difficult matter in connexion with bronchiectasis. In this connexion it is well to remember that most cases of the disease are the result of a non-tuberculous pneumonia in childhood, and that there is no great tendency of the diseased lung to become infected with tuberculosis. A few cases, however, are the result of pulmonary thrombosis due to tuberculosis. Such arise most commonly where the disease has first attacked the pleura. The history of the case, and the presence or absence of signs of tuberculosis in the other lung or signs of

importance. Repeated examinations of the sputum should be made in doubtful cases.

Prognosis.—In slight cases there seems no doubt that complete resolution may occur and the symptoms altogether disappear. More often, however, the condition of the lung steadily becomes worse until an advanced condition of fibrosis occurs. In this case the patient will remain delicate throughout childhood, and will never acquire a robust constitution in later life. As it occurs in childhood, in these cases death is usually due either to an acute attack of bronchitis or pneumonia, or to intracranial abscess. In tuberculous cases the outlook is worse than in the commoner post-pneumonic type. Death is here caused by tuberculous meningitis. Probably, a few of the simple cases become infected with tubercle, but this is certainly not common.

Treatment.—Care must be taken to prevent attacks of acute bronchitis. These may be avoided by the mode of life adopted by the child. Preferably the patient should reside in a warm, dry climate at all events during the winter months of the year.

An effort should be made to disinfest the cavities in the lungs, and to prevent the retention of sputum within them. Creosote, in the form of inhalations (p. 254), or given internally, may be used for the first purpose. Drainage of the infected cavities may be assisted by ordering the child to be inverted every morning. The feet are held up, and while in this position the child is encouraged to cough over a basin, so that the fluid that has accumulated during sleep is evacuated. The general health of the child may be bettered by the administration of cod-liver oil and tonics. The cough may require special treatment, and for this small doses of sedative drugs, such as heroin, are of most use.

For reasons mentioned under the morbid anatomy of the disease, surgical measures are not likely to be of avail in children. Even where the physical signs in the lungs point to one large cavity being present, it is usual to find—should the opportunity for a pathological examination arise—that in place of such a condition we have a collection of small dilated bronchioles.

EMPHYSEMA.

Acute emphysema develops very rapidly in young children. It may be dependent upon the occurrence of bronchitis, pneumonia (particularly consecutive broncho-pneumonia), or pulmonary collapse. In such cases it is compensatory in origin. It may arise chiefly from obstruction to expiration, as in whooping-cough, asthma, and laryngeal stenosis.

Chronic emphysema, producing the barrel-shaped chest and hyper-trophic lungs, as are seen in adults, is not a common condition in

children. When found it is generally due to recurring attacks of asthma, and is associated with more or less chronic bronchitis. In some cases there is, perhaps, a congenital weakness of the pulmonary elastic tissue, for a hereditary tendency may be traceable.

Figs. 58 and 59 show the "old-man chest" in a child of 9 years, with emphysema, chronic bronchitis, and asthma.



FIG. 58.—*Emphysematous Chest in a Child of 9 Years.*

Treatment.—In chronic cases treatment should be directed towards the prevention of acute attacks of bronchitis and asthma, while chronic bronchitis, if present, should be relieved as far as possible (p. 351.)

PULMONARY ABSCESS.

Large abscesses in the lung are occasionally found following pneumonia, most frequently of the primary type, in emaciated children. Small multiple abscesses are much more common, but can hardly be recognised clinically. Usually, the presence of a foreign body in the

terminus of life of its duration extends an abscess. Even large abscesses may be multiple.

Symptomatology.—At the termination of the pneumonia the temperature assumes the hectic type, the child becomes paler and more wasted. The physical signs of this stage are those of an unresolved pneumonia, but there occur further excavation and an



FIG. 102.—Emphysematous Deformity of a Child's Chest.

increase in the leucocytosis. When the pus is developed, the signs are those of an empyema, and the symptoms are closely simulated by that condition. Dislocation of the heart is rare in pulmonary abscess. It is to be remembered that both an abscess and an empyema may be present.

Diagnosis.—This is usually effected by means of an exploring needle, and an operation is undertaken for what is thought to be an empyema. The surgeon is the first as a rule to locate the pus as intrapulmonary.

Prognosis.—The poor condition of the patient, the difficulty in diagnosis, and the fact that more than few abscess is commonly present, all render the outlook in pulmonary abscess very grave. Without operation the disease runs a course of a varying length, and generally causes death. The abscess may rupture into a bronchus and so be evacuated; but this is an uncommon occurrence.

Treatment.—This is surgical, and should be undertaken on the same lines as for an empyema.

PULMONARY GANGRENE.

This is a rare condition, which is of interest in that it is practically the only cause of serious hæmoptysis in young children.

Etiology.—The majority of cases follow pneumonia, usually the consecutive broncho-pneumonic variety. The condition occurs, for the most part, in emaciated children. The acute specific fevers of childhood, notably measles, appear to predispose to gangrenous changes. The presence of a foreign body may set up a similar condition. In rare instances gangrene may supervene upon acute primary tuberculosis. It may follow tracheotomy, or may be associated with cancerous ones. In a few cases it is caused by a septic thrombus in the lung, secondary to some focus of infection elsewhere in the body, as in acute osteomyelitis.

Symptomatology.—The symptoms are rarely characteristic of the disease. In addition to fever and severe constitutional symptoms, there may be hectic of the breath, expectoration of necrotic pieces of lung-tissue (usually due to vomiting), and hæmoptysis. The hæmorrhage may be profuse, even fatal. In young children hæmoptysis of such severity is practically only found in this condition. In infants death usually occurs before much breaking down of the lung is present.

Treatment.—The patient's strength should be supported, as far as possible, by careful nursing and the administration of stimulants. Continuous inhalation of such antiseptics as creosote (p. 154) should be practiced. Any sign of abscess formation is an indication for immediate operation. Where this is practicable but a few cases occur.

ACUTE PNEUMONIA.

The differences between a primary pneumonia, lobar and lobular, and the consecutive or secondary broncho-pneumonia, are fully discussed under the introduction to the latter disease (p. 544). Primary pneumonia is described under the pneumococcal infection (p. 87).

CHRONIC PNEUMONIA.

Interstitial pneumonia, with pulmonary fibrosis, is always accompanied by bronchiectasis, although this may not be recognizable during life. It has been described under the latter heading (p. 352).

ASTHMA.

Much more commonly than is generally recognized asthma begins during childhood, and because it is at the first manifestations of the asthmatic tendency that treatment is of most avail, the disease is one of great importance.

Etiology.—The age-incidence may be mentioned first. Nearly one-third of the cases of asthma originate before the tenth year of life (Salters). Attacks of the adult type are rare before about the eighth year, but it is probable that we can trace the disease during the earlier years of childhood, and even in infancy. An inherited neuropathic tendency is a common and important predisposing factor. Occasionally asthma is itself inherited. Enlarged tonsils and adenoids, any abnormalities at the nasal cavities, and possibly enlarged bronchial glands, predispose towards asthma. Given a suitable subject, the attacks may be set up by bronchitis, indigestion, constipation, and the inhalations of pollen. In older children, hay fever is sometimes seen.

In children the first attack of asthma is, as a rule, associated with bronchitis, and throughout childhood, particularly during infancy, the catarrhal symptoms are more prominent than in adults.

Attacks of asthma may, synchronize or alternate with eczema or arthritis.

Symptomatology.—In infants and young children asthma is rarely diagnosed, and the attack is called one of "bronchial catarrh." It is, however, more than probable that the disease can be recognized during the earliest years of life. The attack takes rather the form of one of bronchitis, but differs from it in that it is set up by some reflex cause, disappears very rapidly, and tends to recur from various trivial causes. Thus, from the eruption of a tooth through a slightly inflamed gum, from some error in diet, from a slight attack of diarrhoea, constipation, vomiting or colic, the infant begins to get short of breath and wheezy. The temperature is a little raised, and the lungs on examination show poor air-entry, and the presence of rhonchi. Sometimes the symptoms are more alarming: the child becomes cyanosed and prostrated, and is thought to be at the onset of a severe attack of bronchitis. The chest is full of high-pitched sibilant rhonchi. With the administration of an aperient the bowels act, and at twenty-four hours, often in fewer, the child is practically well. There can be little doubt that in many cases such an illness as this is asthmatic in type.

Perhaps asthma can be more easily recognized in another form. In young children and infants, particularly those who are rickety and have enlarged tonsils and adenoids, there are frequent attacks of bronchitis which are in no way remarkable. During or immediately following one of these, however, there occurs a definite attack of dyspnoea, which may recur for several nights in succession.

In older children asthma closely simulates the disease as it is seen in adults, and requires no special description.

Diagnosis.—In cases occurring from trivial causes during infancy, the diagnosis can hardly be made in the absence of a history of previous attacks, until the rapid subsidence of the symptoms is seen. In older children, and in some cases in younger children with bronchitis, the symptoms are sufficiently clear to make the diagnosis easy.

Prognosis.—Only a minority of the cases occurring in infants and young children grow into chronic cases of asthma. Most cases in children recover by the age of puberty. As a rule, where there are definite underlying conditions to account for the asthma, such as rickets, enlarged tonsils, adenoids and the like, the results of treatment are good. In older children the same applies. The longer the asthmatic attacks have been present, and the stronger the neuropathic inheritance, the worse is the outlook. Frequent attacks of asthma lead to emphysema and bronchitis. Chronic bronchitis tends towards persistence of the asthmatic habit.

An asthmatic attack is rarely fatal, but may cause death by asphyxia in infants.

Treatment.—We have first to consider the treatment of the asthmatic attack, secondly, the measures to be adopted to break the asthmatic habit.

When an attack is pending, the most valuable drugs are potassium iodide and belladonna. In place of the latter stramonium or lobelia is preferred by some. Antipyrin is also of use. With these an aperient should be given. When the attack is fully developed, the dose of the belladonna should be increased or atropine given hypodermically, together with, in severe cases, a small dose of morphia. Other inhalations of the fumes of various asthma powders work well. Such a powder may be made of equal parts of nitre, powdered stramonium leaves and lobelia, but these inhalations, if used often, undoubtedly have a bad influence in increasing the tendency to further attacks.

In the cases in infants associated with teething and dyspepsia, a simple saline aperient is usually sufficient treatment.

We now have to consider what may be done to break the asthmatic habit. As is well known, the longer a patient has suffered from asthma, the more difficult it is to cure the disease. It is, then, of the greatest importance to endeavour to attack the habit of asthma as early as possible.

In young children a permanent cure is usually effected by the treatment of the conditions predisposing to asthma—namely, rickets and nasopharyngeal obstruction.

In older children we have first to consider their necessary mode of life. In the asthmatic we have a patient in whom normal external stimuli set up an abnormal response owing to the hypersensitiveness of various mucous membranes, most commonly of the nose, bronchi, or stomach. We can approach the matter in two ways. We may so place the patient that nearly all possible stimuli are withdrawn, or we may endeavor so to improve the patient's health that no longer is the abnormal response, the asthmatic attack, elicited. The first method a "coddling" process, is the one invariably adopted in the domestic circle, but it is nevertheless wrong, for it cripples the patient's life without being entirely successful. We have, then, to adopt a sane "hardening" process. The nasopharynx must be set right, enlarged tonsils or adenoids removed, a deflected septum attended to. Hyperæsthetic areas on the nasal mucosa should be sought for, and if found should be cauterized or painted with a 2 per cent solution of silver nitrate. It is, however, important to note that the cauterization of a nasal mucosa which appears quite normal will often get rid of asthmatic tendencies altogether. Where there is a persistent chronic bronchitis and a tendency to superimposed attacks of acute bronchitis, nothing is of so great benefit as a change of climate.

No one place suits all asthmatics, and it is always difficult to say without experiment where a particular case should be sent. As a rule a dry and sunny climate, such as may be found in many south coast towns, is of most benefit. When a suitable climate is found, the child should be gradually accustomed to lead a healthy out-of-doors life. In the more purely sporadic cases a change of room or house may lead to a cessation of the asthmatic attacks.

Again, in some cases particular care must be taken of the digestive tract: the food must be simple and nourishing, and taken at regular intervals. Over-eating and constipation must be avoided.

Lastly, there is the important treatment by tonics, particularly, in view of the neurotic basis of asthma, the treatment by nerve tonics. Arsenic is here a most valuable drug, and may be given, with intervals, for many months.

VI.—DISEASES OF THE PLEURA.

In the great majority of cases acute inflammation of the pleura is referable to a pneumococcal, tuberculous, or rheumatic infection. Some, however, are due to pyogenic organisms other than the pneumococcus, while in a few instances pleurisy appears to be the result

of Hill, when its relationship to the infections already mentioned is doubtful.

The reader may therefore be referred: for Pneumococcal Pleurisy, to p. 100; for Tuberculous Pleurisy, to p. 124, and for Rheumatic Pleurisy, to p. 152.

DRY PLEURISY.

This is usually due to tuberculosis, or, if associated with pneumonia or leucitis, to a pneumococcal infection. Occasionally, it is the cause of pleural pain, usually left-sided, in cases of rheumatic pericarditis. It may seemingly occur as the result of exposure to cold; some such cases are probably tuberculous, but many slight attacks are more probably due to the pneumococcus or allied organism.

PLEURISY WITH SEROUS EFFUSION.

The tuberculous cases form the great majority of the instances of this type of pleurisy. A clear pleural effusion is sometimes seen in rheumatic cases associated with pericarditis, and also in connection with typhoids and scarlatina. In the last, however, excepting in cases of cerebral dropsy, a purulent effusion is much more commonly found.

Occasionally in pneumonia a clear effusion containing pneumococci is found, but as a rule this rapidly becomes purulent.

Speaking generally, then a pleurisy with serous effusion indicates a tuberculous infection.

EMPHYEMA.

In the vast majority of cases an *empyema* is secondary to pneumonia, and is particularly common as a sequel to or associated with the primary type of pneumonia. It is most commonly due to the pneumococcus. In other cases it may be due to septicaemia, appendicitis, subphrenic abscess, or injuries to the chest. Acute osteomyelitis and scarlatina are not uncommon causes of the condition. A few cases are due to a primary infection of the pleura by the pneumococcus.

Mention is sometimes made of a tuberculous *empyema*. This is, however, inaccurate. Tubercle bacilli are occasionally present in an *empyema*, but only a superadded infection by a pyogenic organism can cause a purulent pleural effusion in tuberculosis.

The various organisms other than the pneumococcus, and their clinical significance, have been mentioned in the aetiology and prognosis of pneumococcal *empyema*. *Empyema* is fully described under the pneumococcal infection (p. 101).

SECTION VII.

DISEASES OF THE CIRCULATORY SYSTEM.

I.—CONGENITAL CARDIAC ANOMALIES.

Etiology.—Congenital heart disease, as it is generally called, falls etiologically into two groups: one in which it is due to simple malformation, and another in which fetal endocarditis has played a part in producing the abnormal condition of the heart.

Simple malformation accounts for the majority of the cases of congenital cardiac anomaly, and as a rule is due to arrest of the developmental processes in the heart at an early date in fetal life. This arrest may occur before the differentiation of the various cardiac chambers and of the larger blood-vessels has taken place, but more commonly such differentiation is not absent but is incomplete.

It is to be noted that the development of the heart is almost as complete by the end of the second month of fetal life as at the end of the period of pregnancy.

Under the heading of malformation may be classed the cases of dextrocardia, with which may be associated transposition of other viscera.

Mongolian idocy, as was pointed out by Dr. A. E. Garrod, is frequently associated with improper development of the heart.

Fetal endocarditis can only very rarely be traced as the cause of congenital heart disease. When it occurs, the right side of the heart is usually affected, and most frequently the pulmonary valve segments are diseased. From such endocarditis, arising before the end of the second month of fetal life, arrest of development of the ventricular septum may occur.

It has been said that a family history of acute rheumatism predisposes to congenital heart disease.

Lesions.—In a large number of cases the maldevelopment of the heart is so serious as to be almost incompatible with life. Where death occurs very shortly after birth, a number of serious abnormalities may be found, but as they cannot be recognized clinically, there is no need to discuss them in detail.

In cases which survive the first few months of life the most common condition found shows both pulmonary stenosis and patent septum

ventriculorum. Any single maldevelopment is comparatively uncommon. In addition to these lesions we have to consider aortic stenosis, patent ductus arteriosus, and patent foramen ovale. Triangular or oval lesions are infinitely less common than those which have been mentioned.

Symptomatology.—During infancy the symptoms referable to congenital heart disease are rarely well marked; nor are cardiac bruits always present. It is exceptional to find cyanosis permanently present in these infants. More commonly it is noticeable only after attacks of crying. Often indeed it is but by a routine examination of the chest that evidence of heart disease is found. As a rule these children do not thrive well, they are apt to become unduly cyanosed on crying or on exposure, and they may have attacks resembling faints. Where no test is audible over the heart, such symptoms as these may be suggestive of the correct diagnosis, but often congenital heart disease is found in autopsies upon wasted infants where no such lesion has been suspected during life. Death not uncommonly occurs suddenly in a syncopal attack, or may be due to convulsions, marasmus and hypoxemia.

During the later years of childhood, should the patient survive, congenital heart disease is usually much more easily recognized. There is generally a clear history to the effect that the child has not been able to run about and play as other children do, and that on exertion it becomes breathless and cyanosed. Such children as a rule are undernourished; they feel the cold acutely, and are subject to chills. They are usually very affectionate children, but are excitable and passionate. They are not particularly apt to develop epilepsy at a late age.

The most important symptom is that of cyanosis. This may be deep and persistent—hence the term *morbus caeruleus*—in which case it usually indicates pulmonary stenosis. In other cases it is only transient, and in many is entirely absent. It is accompanied by a compensatory increase in the number of red corpuscles, and of the percentage of hemoglobin in the blood. The exact mode of origin of the cyanosis is not yet fully understood. An intermixture of venous and arterial blood is not necessary for its production, as was shown by Sillé; nor will venous congestion account for all cases. If we hold, as Dr. Loe suggests, that cyanosis "simply means deficient aeration of blood, and that the amount of cyanosis is a measure of the amount to which aeration of the blood has been hindered," we see that it is possible for intermixture of venous and arterial blood to occur without causing cyanosis, and that cyanosis may occur in the absence of any such intermixture, or of venous congestion.

Clubbing of the fingers, toes, and tip of the nose, is present in most cases which show cyanosis, and is probably to be regarded as due to venous congestion (Fig. 66). The clubbing is proportionate to the

cyanosis, and both tend to become more marked as the child's age increases. They usually indicate pulmonary stenosis.

Physical Signs in the heart, although sometimes absent during infancy, are usually conspicuous later. While, however, they enable a diagnosis of congenital heart disease to be made, they do not in practice always provide a certain means of differentiating the type of lesion present in the heart.

Enlargement of the heart to the right is usually present, but it is not generally very marked. Any pronounced bulging is exceptional.



Fig. 855. CONGENITAL (Bicuspid AORTIC) STENOSIS WITH BULGING (CLIPPING BY THE LINDERS).

The area of deep cardiac dullness may be found to be abnormally circular in outline.

The murmur of congenital cardiac disease is usually best heard down the left border of the sternum. It is systolic in time, loud, rough, and often audible over the back of the chest and in the upper parts of the axilla. A fine systolic thrill can usually be felt over the pulmonary area. A second sound is generally audible here, but may be only conducted from the aorta.

On the other hand the heart may be very soft and blowing, and unaccompanied by any thrill. Complete absence of any murmur is rare in cases of congenital heart disease which show any symptoms of that condition. A heart may, however, disappear during a period of illness

from interference of the heart's action. A patent foramen ovale existing alone does not give rise to any bruit. Such a condition is, however, rarely recognizable during life.

Pulmonary Stenosis.—The systolic bruit is here usually rough and loud, and has its point of maximum intensity at the pulmonary area of the heart. It is commonly accompanied by a thrill, and is followed by a loud sound which is not, however, the pulmonary second sound, but the conducted aortic sound. Both cyanosis and clubbing of the fingers are usually present after the age of infancy.

Patent Septum Ventriculorum.—The bruit here is usually softer than in the preceding condition, and is heard at a lower level on the chest-wall. Its area of maximum intensity is in the third or fourth left interspace, close to the margin of the sternum. Both cyanosis and clubbing are absent.

Pulmonary Stenosis and Patent Septum Ventriculorum.—In most cases of congenital heart disease both these lesions are present. We are here, therefore, dealing with the commonest type of case. The systolic bruit is usually best heard in the third or fourth left interspace (suggesting a patent septum ventriculorum), but occasionally two zones of maximal intensity are recognizable, one in the second space and one at a lower level. A thrill is present. The presence of pulmonary stenosis is suggested by the presence or history of cyanosis.

Patent Ductus Arteriosus.—This defect is generally associated with other abnormalities, but in those rare instances in which it exists alone, it may be recognized by a peculiar humming sound, waxing and waning, but continued throughout the cardiac cycle. This is heard loudest over the base of the heart, but it is audible all over the chest. There may be a narrow area of dullness extending vertically upwards along the left border of the sternum and an upward extension of the heart's shadow as seen in a skiagram. A patent ductus arteriosus existing alone, although it produces such conspicuous physical signs, is associated with very few cardiac symptoms and practically does not tend to shorten the patient's life. Its recognition is therefore of some importance.

A diastolic murmur in congenital heart disease is usually due to a patent ductus arteriosus.

Aortic Stenosis.—This rare lesion, due to fusion of the aortic cusps, gives rise to the same signs as the similar acquired condition.

Patent Foramen Ovale.—This is a condition which can hardly be recognized during life. It rarely, if ever, gives rise to a bruit. The foramen closes normally at a varying period after death, and at autopsies on young infants it is often difficult to determine if a patency of the foramen has been of any consequence. A small opening is very frequently found in infants' hearts.

Diagnosis.—From acquired heart disease the congenital cases are

as a rule easily differentiated by the history, cyanosis, type and conduct of the cardiac beats, and by the absence of rheumatic manifestations. The area of maximal intensity of the murmur should be most carefully noted.

Cardiac murmurs heard in children under the age of two years are almost invariably the result of congenital heart disease. Rheumatic endocarditis hardly occurs at such an age, while postinfectious endocarditis is very rarely the cause of a condition which at this age would simulate congenital heart disease.

The loud pulmonary systolic murmur heard in some cases of rheumatic heart disease, and probably due to a dilated cone pulmonalis, sometimes suggests a congenital lesion. It is not, however, accompanied by a thrill, and can with care easily be differentiated.

Of the type of lesion the diagnosis is, as a rule, difficult. The points suggestive of the various most important lesions have been detailed above.

Prognosis.—In addition to the large group of cases which die shortly after birth, a great number die during the first five years of life. According to Holt, 60 per cent of children with congenital heart disease die before they reach the age of five years, while of these one-half die within two months of birth. Only about 5 per cent of all cases survive until the age of thirty.

In the prognosis of any individual case, apart from the kind of life which the patient's circumstances will compel him to lead, the most important point is the amount of incapacity which the heart condition causes. The cyanosis is not an accurate guide in prognosis, for, although where it is very intense it is a bad sign, many cases showing it to a moderate or even severe degree may do well, while those in which there is little or no cyanosis may die young. The best guide is the amount of work and exertion which the heart will stand without breaking down. Where there have been numerous attacks of heart failure, the outlook is necessarily extremely bad.

The prognosis as regards life in cases of patency of the ductus arteriosus existing without any other lesion is quite good.

Any acquired diseases of the lungs or heart are of necessity extremely dangerous to the subjects of congenital heart disease.

The influence of congenital malformations of the heart upon the nutrition of the patient during infancy has already been mentioned. Later in life growth is very apt to be retarded.

Treatment.—This must be symptomatic. Regulation of the patient's life, avoidance of cold, respiratory diseases, and over-exertion, and the administration of tonics, are important points. Where symptoms of cardiac failure arise they must be treated on ordinary lines.

II.—ACQUIRED HEART DISEASE.

Etiology.—Acute rheumatism is not only the one great cause of permanent heart disease, but accounts for the majority of cases of transient heart trouble in children. That this is so is usually clearly evident clinically. Preceding or coexistent symptoms of rheumatism can, as a rule, be accurately traced, but in a few instances, although the heart disease corresponds to the rheumatic type, we are not able to find any evidence of this infection. That this should be so is only to be expected. When we consider how often the slightest form of rheumatism may pass unnoticed by parents, and even by medical men, and how indefinite the signs of the infection may be, even where the heart is severely attacked, it is small wonder that proof of the rheumatic origin of all cases of rheumatic heart disease is not forthcoming.

We must therefore make it a rule to suspect rheumatism first as the cause in any case of heart disease in a child. It is a matter of common experience that when an acquired cardiac affection is diagnosed as non-rheumatic, the diagnosis is eventually proved to be inaccurate.

Other infections may, however, give rise to heart disease in children. These have been considered, where necessary, elsewhere, and here references only need be given.

Acute Myocarditis may be due to rheumatism (p. 350), diphtheria (p. 242), influenza (p. 200), and occasionally to other infections, of which typhoid fever must be mentioned. The pneumococcus produces comparatively little myocardial inflammatory change, except where purulent pericarditis is present (p. 410).

Acute Non-purulent Pericarditis may be due to rheumatism (p. 373), or to tuberculosis (p. 410).

Acute Purulent Pericarditis is, in children, in the great majority of cases, caused by the pneumococcus (p. 107). Occasionally it may be produced by a streptococcal infection and, where secondary to acute osteomyelitis, may be of staphylococcal origin. These other forms, however, differ in no direct way from the pneumococcal type, and to the description of pneumococcal pericarditis the reader is referred (pp. 107 to 109).

Acute Endocarditis.—This, in an overwhelming majority of cases, is due to the rheumatic infection (p. 370). A few cases are due to the pneumococcus (p. 100) and other pyogenic organisms. Tuberculosis (p. 138) rarely gives rise to an endocarditis of any clinical interest.

Malignant Endocarditis is described on p. 386.

1.—RHEUMATIC HEART DISEASE.

Introductory.—It is for its convenience rather than for its accuracy that the traditional separation of the consideration of rheumatic

heart disease from that of the other manifestations of the rheumatic infection is here followed. By adopting this method of description we are in danger of losing sight of that connection between acute rheumatism, acute cardiac rheumatism, and chronic valvular disease, which is so especially close in children.

In approaching the subject of rheumatic heart disease in children, one of the first points needing emphasis is that cardiac symptoms almost invariably mean active cardiac rheumatism, even in the presence of heart disease of old standing. In other words, a cardiac breakdown in a child is practically never due to mechanical causes alone, but to recent changes in the heart superadded to those already there existing. The truth of this statement can as a rule be proved by clinical observation, and the onset of cardiac symptoms can be traced as associated with the development of toxicities, pains, chills, or nodules. In some cases a rise of temperature is the only evidence present of an acute process. Pathologically, signs of recent active rheumatism are almost invariably found in the heart, although a macroscopical scrutiny of the myocardium may be required to substantiate this.

As the result of this fact, we find that in children the clinical picture of heart disease is usually one of an acute rheumatic infection, the child being flushed, feverish, emotional, and highly irritable. Only occasionally do we see the facies typical of chronic valvular disease, such as is so common in adults with mitral or aortic disease.

It is, then, this close association between active rheumatism and heart disease, even in its advanced forms, which is so particularly marked in children, that is apt to be overlooked when the descriptions of the two conditions are separated. But I hope that by this early emphasis of the fact the reader will not fail to appreciate its importance, touching, as it does, all aspects of heart disease as seen in early life.

The second point preliminary to the study of rheumatic heart disease in children, namely, that this is the type which includes the vast majority of all cardiac cases in childhood, has already been mentioned.

Rheumatic heart disease is very rarely found in children under two years of age, and is uncommon in those under five years. Apart from this, which depends merely upon the age-incidence of the infection, the younger the child acquiring rheumatism the more likely is the heart to sustain serious damage. Rheumatism of the adult type, with much joint trouble, high fever, and little cardiac mischief, is rarely seen except in older children.

The great tendency towards recurrences of cardiac rheumatism during childhood has always to be borne in mind.

Lastly, given the rheumatic infection, a great predisposing cause to the occurrence of cardiac trouble is lack of rest to the heart.

ACUTE RHEUMATIC MYOCARDITIS.

The great importance of disease of the cardiac muscle, as opposed to that of the valves or pericardium, has perhaps hardly been sufficiently recognized in the past. To it may be attributed practically all the symptoms of heart disease, in the slightest and in the most severe acute cases alike. It is therefore worthy of extremely careful study, and will here be considered first.

Cases of myocarditis may be conveniently discussed in three groups, according to the severity of the inflammation.

1. We may take first the very slight cases, serious only because they may be overlooked. The recognition of such cases as these is of great importance. The patient may be brought to the doctor for any of a long list of minor ailments. There may be symptoms which definitely suggest rheumatism—sore throat, anemia, pains in the limbs, trunk, neck, or head. On the other hand, the symptoms may be of a nervous type—fidgetiness, irritability, emotionalism, and headache—in which the earliest symptoms of chorea are recognizable. They may be of a kind less obviously connected with chorea, but nevertheless secondary to a state of nervous instability induced by rheumatism; such as acquired nocturnal enuresis, night terrors, screaming, habit spasm, hysteric diarrhoea, and morbid fears. A full description of this type of case is given under *LATENT CHOREA* (p. 105). Sometimes cardiac symptoms are those complained of; the most common is, that the child is languid, or that the excitement of play is so tiring that the child seems utterly exhausted, pale, and ill after it. Others, with none of the excitability which is part of the choreic mental instability, are described as always tired and sleepy. Its feet, the child may say, seem too heavy to lift. Epistaxis is not uncommon in the early stages of cardiac dilatation. The patient's cheeks show a peculiar pink flush. There is a slight rise of temperature at night.

On examination the heart is found to be dilated, an inch, perhaps, to right and left; the heart-sounds are rapid, the first apical sound is short, rather slipping, and very frequently reduplicated. The pulse-rate is rapid, and on the slightest exertion becomes considerably so. There may be some irregularity of the heart.

It is important to notice that on auscultation no apical murmur is heard in the great majority of these cases.

Slight myocarditis may accompany rheumatism of all grades of severity, and is probably present in most, if not all, cases of an active rheumatic infection. If its possibility be borne in mind, the diagnosis of this type of case is easily made. It depends upon the recognition of the earliest symptoms of rheumatism, which has been elsewhere discussed.

2. Myocarditis of a more severe type usually accompanies chronic acute rheumatism or chorea. In this group the heart is dilated one or two inches to right and left; the pulse-rate is increased; and on

auscultation a short systolic bruit is very commonly audible at the apex. It is, however, as a rule localized, or not conducted far into the axilla.

Here mention must be made of cases in which the apical systolic bruit is loud and conducted towards the axilla, such a murmur is said to be "organic." Nevertheless, in the course of a few weeks or months such a bruit may disappear entirely. Where this happens, it is evident that although perhaps the mitral valve has been attacked to some extent, the mitral insufficiency was due to the myocardial change rather than to the endocarditis. Such cases as these must be borne in mind when an attempt is made to give a prognosis on a case of simple mitral regurgitation of recent origin in a child (p. 381).

5. Myocarditis of the severest type is usually associated with pericarditis and endocarditis, the condition of the heart being then known as pancarditis. The clinical picture, which is so often spoken of as typical of pericarditis, is in fact due to, and characteristic of, very severe myocarditis. In such cases the child is intensely dyspnoeic, deathly pale, with cyanosed lips. It may be delirious. Orthopnoea is the rule. The pupils are widely dilated. Severe vomiting may be present. Oedema of the lungs, enlargement and tenderness of the liver, may develop, and occasionally general dropsy and slight jaundice supervene in the worst cases. The heart is enormously dilated, and may stretch from the right nipple into the left axilla. The deep cardiac dullness may reach to the second rib close to the sternum at its upper limit, and occasionally even higher. The pulse is rapid and very feeble. Signs of compression of the lower lobe of the left lung may be present. With slight exertion, such as that of sitting up in bed, sudden death may occur.

Morbid Anatomy.—Comparatively little can be learnt of the myocardial changes from a macroscopic examination alone. The muscle may appear pale, and perhaps slightly greyish. There is, of course, much enlargement of the heart, and usually there are evidences of valvular and pericardial inflammation.

The histological changes* are of great importance, and are in a role very characteristic. There is a little change in the parasympathetic cells, most marked in the most vascular parts of the cardiac muscle, namely close to the endocardium and pericardium. The papillary muscles are much affected. This change is probably for the most part due to a toxicæmia. Principally collections of leucocytes, mainly polymorphonuclear, are seen, and are most likely chiefly the result of oedema due to venous congestion.

Of great interest are certain nodules found in the stroma. These were first discovered by Aschoff, and have been re-described by Dr.

* This account is founded upon Dr. Carey Coombs' article in the *Quart. Jour. of Med.*, Vol. 34, No. 5.



Fig. 46.—Rheumatic Myocarditis. A, Large cells grouped together in numerous subsidiary nodules. B, Smaller celled infiltrate at periphery of nodule. C, Arrow points to dense eosinophilic red, tissue. D, Cardiac muscle. Erypony, 4 Leds; objective, 1 in. Leds.



Fig. 46c.—Rheumatic Myocarditis. This shows the size of the large multinucleated cells (B), which are found in the center of the subsidiary nodules; they are almost as large as the muscle cells (B) lying at the periphery of the nodule. Erypony, 4 Leds; objective, 1 in. Leds.

Carey-Coombs. They consist of large fibroplastic cells, many of which are multinucleate, the nuclei being arranged usually in a chain parallel to the long axis of the cells. These subendocardial nodules are found close to the arterial blood-vessels, and appear to be formed as the response of the tissue to the actual bacteria. Inasmuch as these nodules are regarded as peculiar to the rheumatic infection, they would seem to afford additional evidence of the specific nature of the disease.

In addition, there may be in the myocardium *irritational lesions* remaining as the result of a previous infection.

Prognosis.—In mild cases in which there is either no bruit or one localized to the apex, total recovery is the rule in the absence of severe endo- or pericarditis. The possibility of recovery in those instances in which the bruit is conducted into the axilla has already been mentioned. In connection with valvular disease the prognosis is discussed on p. 381.

In the severe cases the outlook is much worse, but even from most critical conditions partial recovery may occur, although total recovery is exceptional. Of bad import are persistent vomiting, redness, and jaundice, together with great dilatation of the heart. The possibility of sudden death is to be remembered.

Treatment.—Rest is the most important of all therapeutic measures. In acute stages it is imperative; and in severe cases it must be absolute. Its prophylactic value has already been mentioned.

Inasmuch as we are treating an active rheumatic infection in most cases of heart symptoms in children, the drug of greatest value is salicylate of soda. This should be given in doses sufficient to keep the temperature down to normal. It may be well combined with some sodium salt, such as the bicarbonate or citrate, the actions of which have already been explained in this connection, as have also the methods of giving full doses of salicylate (p. 146). Now that the beneficial action of sodium salicylate in acute pericarditis is allowed, it appears unwise to deny its use in rheumatic myocarditis where there is evidence of bacterial activity.

Frequently, however, the condition of the heart is such that symptomatic treatment is necessary, either in addition to or replacing the salicylate. The method of relief of the right heart by leeches has been described under pneumonia (p. 69), and is frequently of the greatest service in rheumatic heart disease. Cardiac stimulants, strychnine, nitroglycerine, caffeine, and brandy, may be used. Subcutaneous injections of camphor in olive oil may be tried if the possibility of an overdose of strychnine is feared. Digitalis is a disappointing drug in the acute stages of rheumatic heart disease of children, and rarely acts very beneficially. The reason for this is that the heart muscle is in them not merely mechanically overstrained, but is the seat of active disease. Restlessness is often allayed by the measures which relieve the right

heart. Opium, however, is of the greatest service in acute heart disease in children, and may be given without risk in the absence of pulmonary oedema. Precordial distress may often be relieved by opium, or by the administration of alkalies which have a tonic action on the cardiac muscle, and possibly reduce the coagulability of the blood. This symptom is often associated with distension within the chambers of the heart, particularly the right atricle. Vomiting, a dangerous and often intractable symptom, is to be treated on the same lines as in adults.

During Convalescence the aim of treatment is to improve the condition of the cardiac muscle, so that it can either recover from its loss of tone or, if necessary, undergo compensatory hypertrophy. To this end cardiac and general tonics may be prescribed, supplemented by the use of a full and nourishing diet. Digitalis is at this stage of distinct benefit. Iron will often be advisable, but, as has been elsewhere emphasized, it is of no use until the activity of infection has entirely ceased. Graduated exercises are of great value, and are easily prescribed for children. The addition of a pillow under the child's head enables him to see about his room, and by the turning of his head he obtains a small amount of exercise. By a second pillow this is considerably increased. Later he may be moved on to a sofa, and then into an arm-chair. Soon he may be allowed to stand, and then to walk for increasing periods of time, and so on until convalescence is complete. Massage is occasionally of use. All forms of excitement must be prohibited, and it must be remembered that these patients are usually highly excitable. Residence in a warm, dry climate, and various measures calculated to ward off further attacks of acute rheumatism, are of value.

ACUTE RHEUMATIC PERICARDITIS.

Symptomatology.—Rheumatic pericarditis is, as a rule, introduced by a stage during which friction is audible, and which may or may not be followed by a stage of effusion into the pericardial cavity. Where, however, there have been repeated attacks of pericarditis, fresh pericardial inflammation unaccompanied by any friction, may arise.

Broadly speaking, pericarditis is associated with the severer forms of the rheumatic infection; thus, the fever is high, the myocarditis is severe and radicarditis is usually present. Where there are many subcutaneous nodules, pericarditis is the rule. Severe chorea may be associated with pericarditis, although, as the cardiac symptoms become grave, the choreic movements usually tend to disappear for the time. Rheumatic hyperpyrexia, a condition now very seldom seen, is usually found in connection with acute pericarditis.

While it is true that pericarditis denotes severe rheumatism, it is equally true that in the majority of the symptoms arise from the associated myocarditis. This is well demonstrated in certain exceptional cases in which the infection of the heart begins in the pericardium,

It is not very rare to find a local pericardial friction in a rheumatic child, whose heart is being examined merely as a matter of routine, and not because of any cardiac symptoms of moment. In such a case percussion will show that the enlargement of the heart is very slight. As the case proceeds, however, the dilatation of the heart increases, owing to the development of further and severe myocarditis; and the symptoms of extreme pulse, cyanosis, dyspnoea, and orthopnoea appear. These, though often spoken of as typical of pericarditis, are thus seen to be due to the affection of the cardiac muscle. Pericarditis is rightly accounted as an extremely serious condition, but its immediate danger is only due to the myocardial change. It



Fig. 63.—Rheumatic Pericarditis: extreme puffiness round the eyes.

is because in pericarditis the muscular changes are almost certain to be very severe, that the appearance of a pericardial friction is to be regarded as a danger-signal.

Most of the symptoms, slight or severe, have therefore been already discussed under myocarditis. Two only require mention here as they are due to the pericarditis itself. Firstly, puffiness round the eyes is very frequently present, and is often a very early and valuable sign. Its appearance in a rheumatic subject should always suggest the possible development of acute pericarditis (Fig. 63). It is generally most marked in the upper eyelids. Secondly, precordial pain is, as has been mentioned, often found in cases of myocarditis, particularly where there is commencing thrombus formation in the chambers of

the right heart; but in pericarditis there may be pain due to the friction. Such is, however, quite exceptional. In some cases the child will seek to obtain relief from it by lying on its back.

Physical Signs.—During the dry stage the physical signs are usually distinctive. A loud, rasping, pericardial rub is heard practically only in rheumatic cases. Tuberculous pericarditis, a comparatively rare condition, seldom gives rise to more than a soft friction; while in persistent cases a rub of any sort is quite exceptional. The friction may often be best heard down the left side of the sternum, but may be localized to any point, or may be universally present over the precordium. It may be soft in quality, but is usually both loud and rough. It may be systolic only in time; more often it is both systolic and diastolic; occasionally its rhythm is triple from the presence of a short auricular friction sound.

It is always important to notice whether the friction is confined to the heart's area alone, or whether it is also produced between the heart and the surrounding structures. The latter condition, known as external pericarditis, is liable to be followed by the development of serious pericardial adhesions. Its presence is shown by a ring of friction round the heart, the sound being modified by the respiratory rhythm. Signs at the base of the left lung, or in the left axilla, are common. Those of compressed lung are simply due to the enlargement of the heart, and not necessarily associated with pericarditis. The development of a pleuritic friction, or of a pleural effusion, is, however, practically confined to those cases of cardiac rheumatism in which the pericardium is involved.

In the stage of pericardial effusion the friction disappears. The signs of effusion are closely simulated by those of severe cardiac dilatation, with which it is always associated in rheumatic cases. In both conditions there are great increase of the deep cardiac dullness, a feeble, rapid pulse, a diffuse, ill-marked apex-beat, and the signs already mentioned at the base of the left lung. The points in favour of the presence of fluid are any marked enlargement of the deep cardiac dullness upwards, the distant character of the heart sounds, and the resistant nature of the dullness over the precordial area. Disappearance of the friction, with no corresponding diminution of the heart, suggests the development of effusion. Signs, such as point clearly to pericardial effusion (as in tuberculous pericarditis), are never seen in rheumatic cases, so that its diagnosis is a matter of the greatest difficulty. It is not, however, of much moment, as the amount of fluid is nearly always small in these cases, and is never in itself a source of danger.

As the effusion disappears the friction may again become audible.

Morbid Anatomy.—Recent pericarditis is usually best seen round the base of the heart. It is shown by the presence of areas of injection

and roughening of the serous surfaces, to which may be adherent soft masses of lymph. Similar changes may be present in any part of the pericardium. Where there have been repeated attacks, the pericardium is thickened and fleshy, and its cavity is seen to be undergoing obliteration by the development of adhesions of varying strengths. Rheumatic nodules, similar to those found in the subcutaneous tissues, are not uncommonly to be felt in the pericardium. In the worst cases, the covering cannot be stripped from off the heart and there are tough adhesions between the pericardium and the surrounding structures. There are the added signs of myocarditis and valvular disease.

In rheumatism, the pericardial effusion may be clear, but is usually slightly turbid, containing flakes of lymph. In a few severe cases it is blood-stained.

Prognosis.—Pericarditis is commonly said to be the most severe form of acute cardiac rheumatism; but, as has been already emphasized, this is because the associated myocarditis is certain to be of a bad type. More accurately, therefore, it may be said that pericarditis occurs only in the severest grade of acute cardiac rheumatism. The immediate prognosis depends upon the condition of the musculature of the heart. Sudden death may occur in pericarditis in either the stage of friction or that of effusion; another fact which shows that the importance of the condition is due to the myocarditis. It is uncommon for a first attack of pericarditis to prove fatal. Recovery, usually partial, but sometimes complete, is the rule. In later attacks the onset of pericarditis is often a terminal event.

The ultimate prognosis depends upon the formation of external adhesions, those merely obliterating the pericardial cavity being of slight moment. The signs of external pericarditis have already been described.

Treatment.—Absolute rest in bed must be insisted upon, and no exertion on the part of the child should be allowed. The possibility of a sudden and fatal syncopeal attack must always be borne in mind. Where possible, the child should be kept lying flat on his back, so that he may not be tempted to move about in watching those around him; but where there is much dyspnoea, he will be more comfortable if propped up on pillows. Restlessness must be relieved as far as possible.

The diet must be light and easily digested. Where there is much dilatation of the right side of the heart, the amount of fluid given may be restricted, and concentrated foods ordered. Of these, Herick's Malted Milk and the flavoured bread of Syntogen are of most use.

For local treatment the procedure is the ice-bag, leeches, and poultices are of use. Application of oil of gaultheria is of no practical value as it is so very slowly absorbed. In one case tested, the anne did not give the salicylate reaction until the fifth day of application.

The ice-bag introduced for this purpose by Dr. Lees is the most beneficial local application, and of its value there is no doubt. In order to obtain the best results, attention should be paid to the following points. The ice-bag should be applied directly to the skin without the intervention of any flannel, except possibly for an hour or two while the child becomes accustomed to its presence. Some form of binder should be arranged to ensure the ice-bag being kept accurately in place. As a rule the binder should be secured in position by straps passing over the shoulders (*Fig. 84*). Over the bag some cotton-wool should be placed, to absorb the moisture which collects on its cold surface. Small pieces of ice should be used to fill the ice-bag,



Fig. 84.—The young Woman in *ARTICLE ON PERICARDITIS* WITH HEART.

which will require to be recharged at least every two hours. As a make-shift, a new mackintosh sponge-bag of the ordinary shape may be used. While ice is being applied, particular attention must be paid to keeping up the warmth of the body by means of hot-water bottles to the legs and feet. The temperature should be taken in the axilla at two-hourly intervals, and should it fall below 100° , it is usually wise to remove the ice for a time.

When an ice-bag which has been properly applied is taken off, an area of hyperæmic skin should be disclosed, resembling that produced by poulticing. Ice is, then, a counter-irritant, and is in fact the best means that we have at our disposal for keeping up constant

counter-irritation. It is probable that the application of cold over the heart also has a direct effect on the inflammatory processes in the pericardium. Thus its use allays the inflammation, reduces the temperature, and tends to quiet the heart.

When the ice-bag is no longer required it should be omitted for gradually lengthened periods of time, until it is discarded entirely.

For drug treatment salicylate of soda is our mainstay. Its value in pericarditis, as was taught for so many years by Dr. Leen, is now generally recognized. It should be given in doses sufficient to control the temperature. Should larger doses than are generally used be required, they should be given as directed on page 456.

The symptomatic treatment of the heart by sedatives and stimulants will generally be necessary. These have already been mentioned in the treatment of myocarditis (p. 477).

One other line of treatment seems of undoubted value in dealing with pericarditis, namely, keeping the child sufficiently under the effect of opium to ensure complete rest. This is not devoid of danger when symptoms of right heart failure are present; but should there be restlessness after the right heart has been relieved, opium morphia should be given.

In rheumatic cases aspiration of the pericardial effusion is practically never necessary. Occasionally pleural effusion, usually left-sided, will require to be removed.

Convalescence should be conducted on the lines laid down under the late treatment of myocarditis (p. 573).

ADHERENT PERICARDIUM.

This is the most serious form of chronic rheumatic heart disease found in children. Not only is the heart embarrassed, both in the acute and chronic stages, by the presence of adhesions, but when they are universal no room is left for the heart to grow. Thus it cannot hypertrophy to compensate for valvular disease, nor can it increase in size as the child develops.

By the term adherent pericardium is meant not merely a condition of adhesions between the two layers of the pericardium, a matter of small moment, but one in which there are in addition adhesions between the pericardium and the sternum, lungs, diaphragm, and mediastinal structures. It originates in repeated attacks of acute inflammation in which external pericarditis has been present.

Physical Signs.—These do not differ from those seen in the same condition in adults, except that precordial bulging is more constant and more marked. There is evidence, therefore, of great enlargement of the heart due to past disease of the myocardium and valves, as well as to the presence of the adhesions. Occasionally, however, the enlargement of the heart is only very slightly marked; but such cases are very exceptional. The adhesions may be universal, or may

be limited to the anterior or posterior surfaces of the pericardium, and thus the physical signs may differ in different cases. Of these, the most important are: fixation of the apex-beat—systolic retraction of the interspaces unaffected by the patient's being placed upon his right side; and the posterior diaphragmatic phenomenon described by Sir John Broadbent, in which systolic intugging is visible in the neighbourhood of the eleventh rib on the left side posteriorly. A short presystolic bruit, and a mid-diastolic bruit at the apex, have been described as due to pericardial adhesions, but these, as will be seen later, may be found in cases of thickening of the mitral valve in the absence of both mitral stenosis and adherent pericardium.

Symptomatology.—The symptoms are those of dilatation of the heart, and are mainly due to embarrassment of the right heart. Even in this condition the onset of symptoms usually means a fresh attack of cardiac rheumatism, rather than a mere mechanical breakdown of compensation. With renewed attacks compensation repeatedly fails and becomes imperfect, and the intervals of comparatively fair health shorten.

Prognosis.—Where the adhesions are universal the outlook is extremely serious. If from no other cause, death is very prone to occur when there is a necessity for the heart to increase in size to cope with the growth of the child's body. During puberty and adolescence, when physical development is proceeding rapidly, and the patient is perhaps forced to earn his living, the heart is unable to undergo a corresponding growth owing to the strangling adhesions round it. Life is seldom prolonged beyond this period.

Where the adhesions are not universal, their presence adds in varying degree to the danger of the valvular and myocardial disease present.

Treatment.—Where adherent pericardium is present, the treatment must consist in preventing fresh attacks of rheumatism, in sparing the work of the heart, in putting the patient in the most favourable position to acquire and maintain such compensatory hypertrophy as can develop, and in treating the symptoms of failing compensation as they arise. Recently, operations undertaken to free the heart partially by removing some of the ribs covering it have been practised. It is not possible to think that in this particular condition such operative measures have a great future.

The symptomatic treatment of the heart has already been described (p. 374).

ACUTE RHEUMATIC ENDOCARDITIS.

For the most part the acute stages of endocarditis cannot be recognised clinically, and the symptoms which are present are those of acute myocarditis (p. 363). Acute valvular disease is almost assured in

cardiac rheumatism, when there is pericarditis. The significance of rheumatic nodules in connection with endocarditis has been discussed elsewhere (p. 148).

Endocarditis of the mitral valve is undoubtedly present in many slight, and even indefinite, attacks of the rheumatic infection, but here is usually of less importance than the stretching of the mitral ring from the associated myocarditis.

A word must be said on the use of the terms "organic" and "functional" as applied to apical systolic murmurs. By an "organic" murmur is generally meant one signifying the presence of mitral endocarditis, while the word "functional" is used for each case in which it is thought that the valve itself has escaped damage. If the terms are used strictly in these senses, it is probably correct to describe most apical systolic bruits which persist for any length of time as organic. But by describing a murmur as "organic," it is frequently implied that it will remain permanently; while by describing it as "functional," it is meant that complete recovery will take place. We have, however, no right to assume that a regurgitant bruit, due only to stretching of the mitral ring from myocardial changes, must of necessity disappear; nor is it possible to suppose that in permanent cases the regurgitation is due to valvular disease rather than to a persistent enlargement of the mitral orifice from muscular changes. It is, indeed, very difficult to imagine that slight scarring at the edge of the valve, where mild endocarditis has been present, is entirely responsible for the mitral insufficiency, although it may well be that the irregularity of the edge of the valve tends to increase the intensity of the bruit.

Here, then, is another example of the way in which the importance of rheumatic myocarditis, as opposed to that of endocarditis, is hardly sufficiently recognized.

The question of the possible recognition of active inflammation of the substance of the mitral valve by means of the mid-diastolic apical bruit is discussed under mitral stenosis (p. 382).

Morbid Anatomy.—The order of the frequency with which the valves are attacked by rheumatism is as follows: mitral, aortic, tricuspid, and pulmonary. In fatal cases both the mitral and aortic valves are usually diseased, while the tricuspid segments are not seldom involved. In less severe cases the mitral valve is often the only one attacked.

The method of infection of the valves is a question which is still a subject of controversy. It was formerly held that they became infected by organisms in the blood passing through the chambers of the heart; but the belief is gaining ground that the valves are infected by organisms through the coronary circulation. This latter view appears the much more likely one, and is in all probability the correct explanation. The mitral valve is attacked the most frequently, because being the largest it is likely to obtain the largest number of

organisms. The aortic valves are probably attacked in many cases by a direct spread of the infection from the neighbouring mitral flaps.

Evidence of recent endocarditis is seen in the minute, bead-like vegetations which occur on the valves. On the mitral and tricuspid flaps they are usually seen in a row close to the free border of the valve. On the aortic valves they develop first in the neighbourhood of the *corpora Arantii*. They may occur also on the chorda tendineae. In colour the vegetations are grey or greenish-grey. In addition there are signs of swelling of the valve-segments, and in most cases seen post mortem there is evidence of old endocarditis.

Treatment.—The treatment of acute endocarditis is on the same lines as that of acute myocarditis (p. 372).

CHRONIC VALVULAR DISEASE.

It has already been pointed out that attacks of failure of compensation in the chronic valvular disease of childhood almost invariably denote fresh cardiac rheumatism. The clinical features of this have been described, and it remains for us here to discuss the various forms of compensated valvular disease, mentioning in particular such points as are of interest in connection with them, as they appear in childhood.

Mitral Regurgitation.—This, although the commonest chronic valvular lesion in children, only requires short notice here, as it differs but little from the same condition in adults. In its production two factors are present in most cases, the myocardial and the valvular. Of these, as has been already emphasised, the former is the more important.

A systolic murmur is audible at the apex, and may be localized there, or may be conducted into the axilla and to the angle of the left scapula. With this there are signs of enlargement of the heart. The pulmonary second sound is loud, often reduplicated, and accompanied by a diastolic shock. At the same area a systolic thrill is frequently present. This, which it is to be noted may be quite loud, is probably dependent upon dilatation of the pulmonary cones.

An apical systolic thrill is very frequently present in marked cases of mitral regurgitation in children, and is in fact by far the most common thrill found in young subjects.

Prognosis.—The diagnosis of mitral insufficiency is easy, but it is much less easy to decide whether it is likely to be permanent, or only temporary. Allusion has already been made to this point. Where the lesion is of long-standing, the enlargement of the heart marked and accompanied by much hypertrophy, the murmur loud, rough, replacing the first sound and conducted into the axilla, there is little likelihood of recovery taking place. On the other hand, where the condition is of recent origin, and the signs are those of dilatation rather than of hypertrophy of the heart, and the thrill is short, soft, and localized, there is every hope that a return to the

normal will take place. But between these two groups of cases are many instances in which the outlook is a matter of doubt; and it is to be remembered that recovery not seldom ensues in the course of a few months, or years, in cases in which such an event appeared extremely unlikely. Inasmuch, then, as we cannot recognize at the time how much of the incompetency is due to myocardial disease and how much to valvular disease, it behoves us, in any case which is certainly one of pure mitral regurgitation, to take as hopeful a view as possible. Even where recovery does not occur, a considerable amount of improvement may take place in time as the result of restoration of the function of the cardiac muscle.

Mitral Stenosis.—The first thing to recognize about this form of valvular disease is that it is distinctly uncommon in children, and is found in them practically only during the last years of childhood. It is a condition of which the diagnosis is often made in rather haste.

The rarity of mitral stenosis in children is due to the fact that its development is a lengthy process. Not only have sclerotic changes to occur throughout the substance of the mitral flaps, but the myocardium has to recover sufficiently to allow the mitral ring to approach the normal in size. This is well seen in some cases in which, as the signs of stenosis develop, those of regurgitation disappear.

Development of Mitral Stenosis.—When we examine the valves at a case of mitral stenosis, we see that healing processes have occurred throughout their substance, and from this we may conclude that at one time there has been an inflammatory change in them, not confined to their edges, but diffused throughout the entire valve-segments. That is to say, in the development of mitral stenosis, and preceding the actual narrowing of the mitral orifice, there must have been a stage in which the valve-segments were swollen and stiffened by inflammation, which later gives rise to sclerotic changes throughout the valve.

The question, therefore, arises, Are we able to recognize directly this inflammatory stage, and thus to forecast and to trace the development of true mitral stenosis? It is held by many that by means of the presystolic-reduplication of the second apical sound, and by the mid-diastolic bruit at the apex, we are enabled to diagnose such an inflammatory thickening of the mitral valve as will later develop into true stenosis.

The stages in the development of mitral stenosis may be characterized thus:—(1) The second apical sound shows presystolic-reduplication; (2) The second half of the second sound lengthens into a short bruit, the mid-diastolic murmur; (3) The mid-diastolic murmur lengthens until it comes to occupy the greater part of diastole, and tends to end in a crescendo murmur leading up to a rather loud first sound; (4) The early part of the murmur disappears, and only a short, rather indefinite, presystolic bruit remains, accompanied by a very short thrill and ending in a loud first sound; (5) The presystolic murmur lengthens,

becomes rougher, and more crescentic in character, is accompanied by a rough thrill, and ends in a very loud and slapping first sound. It is only in the presence of the signs given under the last heading that we can be tolerably certain that actual mitral stenosis is present, the previous stages representing merely such a thickening of the valve as will in time lead to stenosis, or, in a word, representing stenosing rather than stenosis.

These stages are represented graphically in Fig. 65.

The *pansystolic* reduplication of the second sound is heard over a small area internal to the apex-beat of the heart. It is audible in the same place as is a typical presystolic bruit, and, as it is very strictly localized, it needs to be examined for with care. The character of the reduplication is very different from that so commonly heard in the second

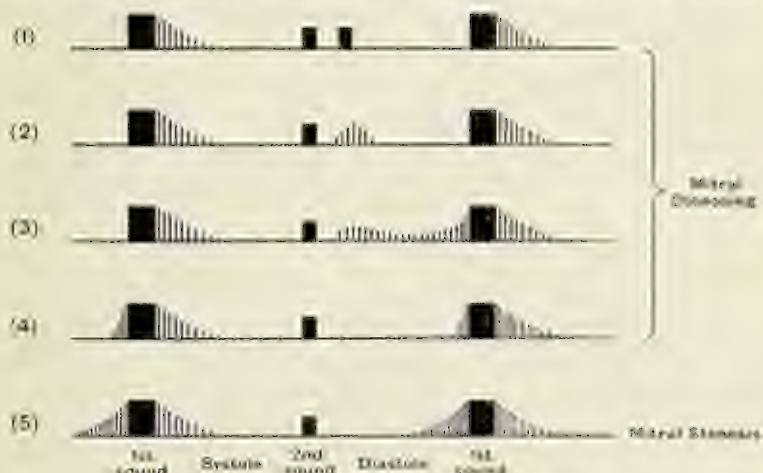


Fig. 65.—Nos. 1-4 show the earlier stages of thickening of the mitral valve as they proceed to stenosis; No. 5, the sign of mitral stenosis.

sound heard, in that it is much more deliberate, the second half of the second (sometimes termed the "third" sound of the heart) being separated by a distinct interval of time from the first half. As the result of this, a characteristic triple rhythm is heard. The explanation of this "third" sound, which has been advanced by Dr. Lees, is that it is due to the tightening of the swollen mitral valve-segments at the last moment of diastole. In normal circumstances this possible mitral sound would synchronize with the second sound, and so be unrecognizable; but when the valve is swollen it is delayed, so becoming audible.

The *mid-diastolic* murmur is heard over the same small area internal to the apex-beat. The exact method of causation of this bruit is a

matter of some difficulty. It is generally supposed that it is caused by thickening of the mitral valve, which prevents the valve from falling away immediately before the blood-stream entering the ventricle.

Dr. Carey Coombs has, however, advanced the theory that the origin of the murmur is myocardial and not valvular, and that it is due to a relative stenosis of the mitral orifice, the ventricle being more greatly dilated than is the mitral ring.

It would seem likely that here, as in the production of a mitral systolic bruit, both the valve and the myocardium may take part, in some cases the valvular factor predominating, in others the myocardial. This would appear to be so, because we see two groups of clinical cases. In one the mid-diastolic murmur is faint, changeable from day to day, being replaced perhaps by a pseudo-reduplication, or even disappearing while the patient is at rest. In such a case, although it is highly probable that the valve is infected, it is unsafe to foretell the later development of mitral stenosis. In the other group of cases showing the mid-diastolic murmur, the bruit is loud and persistent, and in such the ultimate occurrence of mitral stenosis is almost assured.

The other stages which have been enumerated need no further mention.

Just as we can watch the development of mitral stenosis through the various stages which have been described, so, as the result of rest and treatment, we may observe the physical signs of a later stage changing into those of any earlier one: a short presystolic bruit, for instance, becoming typically mid-diastolic.

Aortic Lesions.—While the aortic valves are affected in a large number of cases of rheumatic endocarditis, the condition is not often one of clinical importance. In most cases of aortic endocarditis nothing more is present than a slight roughening of the aortic valves, which gives rise to a systolic aortic murmur. Such should not be classed as cases of aortic stenosis, which is a condition of extreme rarity in childhood.

Aortic regurgitation is comparatively uncommon in children, and when it occurs it is always associated with disease of the mitral valve. It is more common in boys than in girls. In some particulars it differs slightly from the same condition in adults. A typical aortic facies is very rarely seen in children. The four horns of capillary pulsation are not prominent in children with enlargement of the heart, so that in order to be diagnostic of aortic regurgitation, it should be very definitely recognizable. In a child the diastolic murmur of aortic regurgitation is often well heard at the apex of the heart, and sometimes is best audible there. Usually it is heard better over the aortic valve itself on the left side of the sternum than at the aortic area of the heart. When heard at the apex, the aortic diastolic murmur has to be differentiated from the mid-diastolic mitral bruit. It can be distinguished from this by the facts that it is audible in the

enter rather than the inner side of the apex-beat, and is often traceable upwards towards the base of the heart, and that it is not associated with the tripping of the rhythm that is so characteristic of the mitral lesion. In cases of difficulty, the question can usually be settled by examining for the other signs of *aortic regurgitation*. In children, pains that can be termed anginal in character are very uncommon.

In extremely rare instances the signs of *aortic regurgitation*, developing during an attack of cardiac rheumatism, disappear when the activity of the infection ceases.

Tricuspid and Pulmonary Valvular Lesions.—The tricuspid valve is found affected in a considerable proportion of fatal cases of cardiac rheumatism. During life, however, the only condition which is at all commonly seen is that of tricuspid incompetence, due chiefly to myocardial change, and secondary to disease of the left side of the heart. Tricuspid stenosis is a rare result of rheumatic valvulitis.

Pulmonary valvular lesions, due to rheumatism, are so rare as hardly to enter into practical medicine.

Prognosis.—The prognosis in valvular disease of children differs in several ways from that of adults. In the foremost place we must put the ever-present possibility of recurrences of active cardiac rheumatism. This, speaking generally, makes the outlook more serious and more uncertain than in older patients. Again, in the child the heart has to provide for the growth of the body. At such periods as puberty and adolescence great strain is laid upon the heart by the rapid physical development. In hospital cases the patient will begin, as a rule, to support himself by work after the age of fourteen. In children the general nutrition of the body responds rapidly and severely to any form of disease, and under such conditions the nourishment of the cardiac muscle is impaired more quickly in them than in adults. The presence of external pericardial adhesions adds greatly to the work of the heart, and consequently to the danger in valvular disease. On the other hand, children have the advantage over adults in this, that the coronary arteries are very rarely diseased, so that if the child's general condition is good, the heart muscle obtains its full share of nutriment, and consequently has its best chance of undergoing hypertrophy.

The possibility of mistaking myocardial for valvular disease has already been mentioned.

In any given case the prognosis depends, in addition to the points already mentioned, upon the extent of the lesion (best judged by the amount of compensatory hypertrophy which has been necessary), the general nutrition of the child, the home surroundings, and the kind of life to which the patient is destined. The presence of valvular disease increases the danger of all forms of acute illness.

Treatment.—Failure of compensation depends almost always

open fresh cardiac rheumatism; and the treatment of active rheumatism of the heart has already been detailed (p. 372).

While compensation is satisfactorily maintained, the aims of treatment are to keep up the general nutrition, and thus the nutrition of the heart; to prevent overstraining of the heart; and to ward off, as far as possible, recurrences of active rheumatism.

It is to be remembered that, in forbidding the usual athletic exercises to a child, particularly to a school-boy, we are handicapping the child considerably; more especially at a public school. Such forms of athletics as impose a sudden and severe strain upon the heart—for instance, racing and football—are less suitable for children with any cardiac disorder than are cricket, tennis, and fives. Drilling, mild gymnastic exercises, and dancing in moderation, put less strain upon the heart. Where the child is suffering from such heart disease as must prohibit his taking part in the various sports of school life, it is preferable that he should be educated at home, or at some small day-school, rather than at a public school.

In dealing with the children of the poorer classes, patients with heart disease should be certified as unfit for the ordinary school and suitable for education in a school for physically defective children (Appendix B).

2.—MALIGNANT ENDOCARDITIS.

Malignant endocarditis, sometimes badly termed *ulcerative*, *infective* or *septic* endocarditis, is rare during childhood. The infecting organisms are most commonly the pneumococcus, staphylococcus, and various streptococci. Although suppurative conditions—abscesses, empyema, purulent pericarditis and other media—are common in early life, it is only rarely that malignant endocarditis arises from them.

Dr. Forryson has brought forward evidence to show that the rheumatic organism can of itself produce malignant endocarditis, and this view is certainly in accord with experimental results. The whole problem is, however, one of great intricacy. On the one hand are the difficulties concerned with the bacteriology of rheumatism, and on the other the difficulty of defining precisely what is meant by the use of the term "malignant." In most instances, it is true, there are obvious differences between a simple and malignant endocarditis, but there are borderline cases which can hardly be satisfactorily classified by the light of our present knowledge. The trend of modern pathological opinion seems to be towards regarding the essential difference between the two types of endocarditis as due to a difference in the virulence of the infection rather than to a difference in the type of infecting organism. If this be the correct view, it becomes almost inconceivable that the rheumatic agent, which so frequently acts up simple endocarditis, should never give rise to a condition which might properly be termed malignant.

While it is not as yet generally accepted that the rheumatic organism produces a type of malignant endocarditis in man, yet the association between cardiac rheumatism and malignant changes in the valves is close. Not uncommonly the more serious form of disease starts in what appears to be an attack of cardiac rheumatism, while in other cases it is seen that a previous attack of cardiac rheumatism predisposes to ulcerative endocarditis. This association can be explained in various ways. In rheumatism the tonsils are damaged, and thus may be assumed to allow either of a double synchronous infection, or of a pyogenic infection following some length of time after the rheumatic attack. Valves which have been previously damaged by rheumatism are, from their vascularity, particularly liable to infection by any organism circulating in the blood-stream.

Symptomatology.—The early symptoms of malignant endocarditis are often highly difficult to recognize. Perhaps most commonly the heart is thought to be the seat of a rheumatic infection, but the progressive character of the disease, with enlargement of the spleen, prolonged fever, signs of increasing cardiac damage, and progressive emaciation, make the diagnosis of malignant endocarditis probable. While, later, the occurrence of infections, purpura, diarrhoea, vomiting, rigors, and delirium, settles the question of the type of the infection.

In other cases the amount of cardiac damage which can be recognized during life is comparatively small, and the symptoms are chiefly those already enumerated, which can be referred to the severe toxæmia. A remittent fever, grave marion and purpura, with diarrhoea and possibly bleeding from the bowel, may make up the clinical picture of some cases of malignant endocarditis.

Occasionally death is due to exhaustion, but more often it occurs as the result of a cerebral embolism, followed by cerebral hæmorrhage.

Diagnosis.—From what has been said already it will be seen that the recognition of malignant endocarditis is often a matter of great difficulty, while its exclusion is frequently impossible. In cardiac cases, enlargement of the spleen is always a sign which should give rise to the gravest apprehension. In other cases, the recurrence of the symptoms of septicæmia which have been mentioned is very suggestive of malignant endocarditis. A blood-culture may be of use in diagnosis but it is not uncommonly negative, even in malignant cases.

Frequently a diagnosis of malignant endocarditis is erroneously entertained owing simply to a lack of appreciation of how severe and persistent may be the acute processes in rheumatic carditis.

Treatment.—It is doubtful whether recovery ever occurs in this disease in children. Large doses of quinine, and various sera, have

been tried, and occasionally they seem to give temporary benefit. A vaccine prepared from the organism recovered from the patient's blood may be tried.

3.—DISORDERS OF THE CARDIAC RHYTHM.

Irregularity of the heart's action is very common during the years of childhood, and is apt to be found more particularly in nervous, anæmic, and illing children. It may be a sign of diptheritic, rheumatic, or influenza heart disease.

An interesting type, first pointed out to me by Dr. A. E. Garrod is that which follows measles. Acute rheumatism may follow measles, but the cases I am mentioning here have not, I think, their origin in that infection. In them the irregularity of the heart is very marked, and often lasts for several weeks. The condition, as I have seen it, has been devoid of danger, and is benefited rather than rendered worse by a moderate amount of exercise.

Irregularity of the pulse is a well-known but not very useful early sign in tuberculous meningitis.

As in adults, irregularity of the heart may arise from nervous indigestion, cigarette-smoking, tea-drinking, and renal disease.

Tachycardia occurs from most of the conditions enumerated above.

Bradycardia is an uncommon disorder of the heart in children. In its most serious form it is seen in diptheritic heart disease. It is occasionally seen following influenza. It is also a sign of increased intracranial tension, and as such is a more valuable, although later, sign in tuberculous meningitis than is cardiac irregularity. As in adults, it may follow an over-dose of digitalis.

Rheumatic bradycardia is a comparatively rare condition. I have seen it in a few cases of apparently mild rheumatic myocarditis. In them the pulse-rate dropped to fifty per minute, or even slightly lower, but such bradycardia lasted only a few days, and seemed devoid of any particular significance.

Treatment.—The treatment of these cardiac disorders must be directed towards removing their causes where possible. It is often wise to ascertain, by examination, whether the condition is made better or worse by exercise, for this may give a rough guide to the line of treatment to be adopted.

III.—DISEASES OF THE ARTERIES.

Arterial disease is rarely recognizable during childhood, although it is possible that damage to the arteries may be caused by the various infections of children. Scarlatina and acute rheumatism may certainly

produce lesions in the arterial walls, and it is probable that such effects are not confined to these infections. No clinical signs, however, of such damage are seen during the early years of life.

Thickening of the arterial walls of sufficient degree to be recognizable in children during life is most uncommon. It is occasionally met with, and may be due to inherited syphilis, chronic interstitial nephritis, and congenital cystic disease of the kidneys.

Aneurysm in children is almost invariably the result of an embolus, and is very rarely seen at this time of life.

Arterial hypoplasia is a very rare condition, studied by Virchow. In it some of the main arteries are abnormally small. It is hardly to be diagnosed during life.

SECTION VIII.

DISEASES OF THE GENITO-URINARY SYSTEM.

1.—THE URINE IN CHILDHOOD.

Normal Urine.—Some details concerning the normal excretion of urine during the years of infancy and childhood require mention.

Frequency of Micturition.—During the first few months of life urine may be passed as often as twice an hour in the waking hours, while during sleep it may be retained for two or three hours. Between the eighteenth and twenty-fourth months voluntary control of the sphincter of the bladder is acquired, and the child will be able to retain urine for two or three hours while awake, and for from four to six hours during sleep. By the third year the child should be able to sleep for six or eight hours without passing urine.

Daily Excretion.—The amount of urine passed daily by an infant is relatively large. During the first six months, with the exception of the first week of life, from 5 to 16 oz. are passed daily. From the sixth to the twenty-fourth month the output is from 8 to 20 oz. On a diet of sterilized whole milk the excretion of urine is much diminished.

From the third to the twelfth years the amount passed daily may be roughly estimated by multiplying the years of the child's age by 2·5 (Goodhart).

Specific Gravity.—In the first few days of life the specific gravity is high, but it quickly sinks to about 1002 or 1004. At the end of the second year it is about 1010, and from this time onward it gradually increases. At puberty it reaches the adult figure.

Constituents.—During infancy the quantity of urea excreted is very small, while that of ure acid is high. The elimination of ure acid is greater in the new-born than at any later age.

In the first few days of life the urine frequently shows crystals of ure acid, hyaline casts, and a trace of albumin. In nurslings glycosuria is often present.

Albuminuria.—The causes of this condition, which occurs so easily in children, are very numerous. Two types here need mention—the cyclical and paroxysmal.

Cyclical Albuminuria.—This begins to get common in children at about the seventh or eighth year of life, and is most frequently found at the tenth year, or later.

The type of child affected is the neurotic. There are usually rather vague symptoms of ill-health, pale, capricious appetite, dyspepsia, and constipation, while various nervous symptoms, headaches, night-terrors or milder disturbances during sleep, moetal fears and the like, are commonly present. Lack of tone in the involuntary muscles is seen in many ways. Dilatation of the stomach is common, but the vasomotor system is usually most affected. The pulse-tension is low, but varies to an abnormal extent according to the position of the limb—with the hand hanging the tension is markedly greater than with the arm uplifted. The heart is often slightly dilated. The hands are very frequently cyanosed, and show salmon-coloured areas of skin against the blue background. These children are very prone to suffer from chilblains. Epistaxis is of frequent occurrence in them.

Dr. Sutherland has described a group of cases in children as "neurasthenia with cyclical albuminuria," and I have frequently found it in those that I have regarded as being examples of nervous instability due to a slight rheumatic infection (latent chorea). While without doubt these two groups overlap each other, yet I think there is certainly room for both of them in considering the causation of cyclical albuminuria. Sore throats, fidgety movements, and pains in the limbs and sides point towards the rheumatic class, otherwise the symptoms of the two groups are the same. Cyclical albuminuria has also been reported in cases of fully developed and convalescent chorea.

The diagnosis of the cyclic form of albuminuria rests upon the absence of symptoms of nephritis, and of casts in the urine, together with the effect of rest upon the passage of the albumin. With twenty-four hours' rest the urine becomes free of albumin, and as a rule the early morning urine contains none. It appears in its greatest amount at midday, but is still present in the evening.

The prognosis, where organic disease of the kidneys can be definitely excluded, is good, but the condition may last for a long while.

In *treatment* the rheumatic cases are usually benefited by a course of sodium salicylate and alkalies. The mental depression, nervousness, and loss of appetite are often improved, while the pains disappear under such drugs. Apart from this, reliance has to be placed chiefly upon general tonic measures and the administration of strychnine. In all cases, removal from home to a healthy, bracing climate is the most valuable measure, and in many instances, apart from all considerations of climate, it is best to get the child away from its parents, and amongst strangers.

Paroxysmal Albuminuria is probably to be regarded as a sub-group of the cyclic cases. In it albumin appears in the urine for a period of two or three days, and then disappears. During the attack the child may be slightly out of health, but quickly returns to the normal.

Hæmaturia.—There are many cases of hæmaturia in children. In the first few weeks of life hæmaturia may be due to a septic infection,

or to the passage of *uric acid* crystals, but, if associated with the signs of acute nephritis, is generally due to inherited syphilis. Struvy is by far the most frequent cause during the latter half of the first year. From this age onward, until about the fifth year, malignant disease of the kidney is not very uncommon. Purpura and the malignant fevers may give rise to hæmaturia, while hæmophilia is a rare cause.

In addition there are the diseases of the kidneys, bladder, and urethra, similar to those which are common causes of hæmaturia in adults. In children occasionally hæmaturia arises with no apparent cause. Usually in such instances it lasts only a few hours. The passage of uric acid crystals probably explains some of these cases.

Hæmoglobinuria.—This is rare in children. It is described as occurring in the new-born in an epidemic form (Winkler's disease). It is here associated with cyanosis and jaundice, and is usually fatal. *Thrombocythæmia* occurs most frequently in syphilitic children. It is occasionally seen in the infectious fevers, notably scarlatina. Sometimes it seems to follow exposure to cold, and may be associated with Raynaud's disease. It passes off after a few hours. In these peroxysmal attacks it may alternate with albuminuria.

Glycosuria.—This is not uncommonly present in nursing infants, in which case lactose is the sugar passed in the urine. Diabetic glycosuria may occur at any age during childhood. Pentosuria is a very rare but harmless condition, of which all instances up to the present have occurred in Jewish families. Alcapuronic urine (p. 294) will reduce Fehling's solution. Diabetes mellitus is described on p. 50.

Polyuria.—Not uncommonly a mother will complain of this symptom in her child who is probably only suffering from abnormal frequency of micturition. Persistent polyuria is extremely uncommon. It may be noted in cases of diabetes mellitus, diabetes insipidus, and in the rare examples of chronic interstitial nephritis occasionally found in children.

Bacilluria.—Bacilluria is found, if sought for, in a large number of children. It is much more common in girls than in boys. The urine is most frequently infected with the *B. coli* commensals, the condition to which Dr. M. McCrea has given the convenient name of "coluria" (*Practitioner*, 1910).

Cases of coluria may be divided into various clinical groups:—(1) The signs and symptoms of cystitis may be present (*Coli-cystitis*, p. 405); (2) To these may be added those of renal involvement (*Pyelocystitis*, p. 406); (3) Coluria may be the only urinary sign early in the peracute cases described under the title of acute pyelitis (p. 402); (4) There may be no symptoms, either local or general.

(5) In a last group may be placed cases of simple coluria in which

the urine is cloudy from the presence of bacilli and very acid, but contains no pus or cells from the bladder or kidneys. The symptoms here are chiefly referable to the hyperacidity of the urine, and consist of frequency of micturition, often nocturnal, especially at night, and sometimes slight dysuria. To these may be added slight constitutional symptoms which are liable to be overlooked unless the child is under close observation. The most constant of these is a rise of temperature which may only appear at night. In other instances pallor and loss of appetite may be noticed. This condition is to be remembered as a common cause of acquired nocturnal enuresis. The diagnosis is suggested by the symptoms and the cloudy appearance of the urine and confirmed by the recognition of the bacilli in the centrifugized urine or on culture. The treatment consists of rest in bed if the temperature be raised, and the administration of such alkalies as sodium citrate or bicarbonate in large doses until the reaction of the urine is alkaline. In stubborn cases urotropine in 5 or 10 gr. doses may be given in addition.

Pyuria.—Pus is not uncommonly present in the urine of children. It may come from the kidneys; in acute cases from pyelitis, pyelonephritis, or pyonephrosis; in chronic cases from tuberculous disease of the kidney or renal calculus. If from the bladder, it is most commonly due to cystitis set up by the *B. coli* organism, or by vesical calculus. Vaginitis is a very frequent cause of pus being present in the urine, and may lead to mistakes in diagnosis. Occasionally an abscess may open into the urinary tract: such may arise in connection with appendicitis or spinal cancer, or may be perinephritic in origin.

Acetonuria.—A description of the conditions associated with acetonuria and the tests for acetone are given on pages 34 and 35.

Anuria.—In infants, arrest of the urinary secretion is not uncommonly seen, and is probably due to uric acid induration of the kidneys. The urine passed later is highly concentrated, and contains many crystals of uric acid. In older children anuria sometimes occurs, and appears to be of nervous origin. This is not of serious import: it may be treated by warmth, fomentations being placed over the kidneys, and the administration of some citrate of potash.

Suppression of urine is seen in cases of acute nephritis, in severe diarrhoea, and in moribund children.

Dysuria.—Apart from local conditions of the urinary passages, dysuria is most frequently due to the passage of urine which is highly concentrated, abnormally acid, and which may contain crystals of uric acid. Such conditions may be predisposed to by errors of diet and by gastro-intestinal derangement. Hyperacidity is often associated with bacilluria (colibacilia). The treatment consists in increasing the

amount of fluid taken, relieving constipation, and in the administration of alkalis, such as potassium citrate.

Alcaptonuria.—This is a rare condition, occurring almost exclusively in male children, and showing a very strong hereditary tendency. In affected subjects it is present from birth, and is a life-long, incurable and practically harmless condition. The only inconveniences which may be caused by it are that the patient may be suspected of diabetes mellitus, or that in old age some blackening of the cartilaginous structures of the body, strabismus in the eyes, may occur (ochronosis).

The urine of an alcaptonuric becomes dark on standing, first brown, ultimately black. This change is quickened by the addition of alkali to the urine. As a consequence, the napkins of the infant are stained brown, particularly if they have been previously washed with soap. For this state of things the child may come under medical observation. In addition to the darkening with alkalis, the urine reduces Fehling's solution. The best test for alcaptonuria is the production of an evanescent blue coloration on the addition of a few drops of the urine to a dilute solution of ferric chloride. This is pathognomonic.

Alcaptonuria is an "inborn error of metabolism," and probably depends upon the absence of a certain ferment in the liver. For a complete account of the condition, and of the interesting results to which the study of it has led, the reader should refer to Dr. A. E. Garrod's *Croonian Lectures* of 1909.

II—DISEASES OF THE KIDNEYS.

MALFORMATIONS.

Occasionally these are of clinical interest. The kidneys may be fused into an oval mass, or one kidney may be rudimentary or absent, the other being considerably hypertrophied. In such cases as these, especially if there is associated malposition, the tumour may give rise to errors of diagnosis. Most commonly such a tumour is palpable in the region of the prominence of the sacrum. Where one kidney is rudimentary or absent, the suprarenal capsule is usually present and normal. The ureter may be absent, but as a rule it is present though impervious. The hypertrophied kidney is liable to degenerative change in adult life. Supernumerary ureters are of no clinical interest.

Holt states that all malformations of the kidney are twice as common on the left side as on the right, and that males are more often affected than females.

CYSTIC DEGENERATION

of the kidneys may cause very large renal tumours, and may thus

give rise to obstructed labour at the time of birth. In many cases, however, there is comparatively little enlargement of the organs, and as no symptoms are produced which can be recognized as renal in origin, the condition is found accidentally in autopsies, perhaps most often in those on children under one year old. Where less of the kidney substance is involved, as life proceeds arterial changes may develop, and occasionally a thickened renal artery in a child may be due to this cause. Later, uræmic symptoms may develop, or cerebral hæmorrhage may cause death at about the age of twenty.

CONGENITAL HYDRONEPHROSIS.

Hydronephrosis of congenital type is referred to on p. 400.

ACUTE NEPHRITIS.

Acute diffuse nephritis is in the majority of instances secondary to a recognizable infection. There are many infective diseases which may cause an acute inflammation of the kidneys. *Scarlatina* is the most important of these, as it is the most common cause. *Diphtheria* comes next in order of frequency, while measles, varicella, typhoid and influenza originate a few cases. Inherited syphilis is the commonest cause of acute nephritis during the first few months of life. *Pneumonia* is not at all rarely associated with nephritis, especially in certain epidemics. Acute rheumatism, although very commonly causing albuminuria, only rarely gives birth to acute nephritis. Occasionally, however, the onset of the symptoms of nephritis in a rheumatic child synchronizes with the development of rheumatic arthritis or nodules. Purpura, presumably the result of some bacterial infection, is sometimes associated with an acute nephritis, which may be followed by chronic parenchymatous changes. Acute infective diarrhoea may set up an acute renal inflammation, but the *œdema* so often seen in diarrhoeic diseases has rarely a renal origin.

In a smaller group of cases the renal inflammation is apparently primary. Such may arise at any age. It is possible that many of these are pneumococcal in origin. The well-known influence of cold, the co-existence of *inocchia* in many cases, the recognized association between pneumonia and nephritis, in some of which instances pneumococci can be found at the renal, all suggest that a "primary" pneumococcal nephritis is likely to be of fairly common occurrence.

Symptomatology.—Usually the symptoms are definitely indicative of the disease, but occasionally, particularly in infancy, they are obscure, and may lead to a mistaken diagnosis unless an examination of the urine be made. A diminished excretion of urine is the most constant sign of acute renal disease.

It must be remembered that uræmia in children, and especially in infants, is usually associated with a rise of temperature.

As a rule, the onset of the symptoms is after this manner. Following one or two days' malaise and loss of appetite, pallor and larid droopy are noticed, and the urine becomes blood-colored, or smoky, and scanty in amount. Occasionally more severe symptoms, such as persistent vomiting or convulsions, may be present, while in other cases all constitutional symptoms may be absent. The amount of droopy present is very variable, but oedema of the eyelids is almost constant.

In the absence of cardiac dilatation the pulse-tension is high. The heart is rapid, the first sound at the apex double. The "lub-bur" first sound simulating a short presystolic bruit is very commonly heard. The second aortic sound is loud. When dilatation supervenes, the heart may become irregular and the sounds "tic-tac" in character. Bronchitis is frequently present at the onset of the illness.

The nervous symptoms are of interest. As a rule the child is at first very miserable. The strict rest and diet, the hot-closed eyes, and the periodic appearance of the hot-air bath conduce to his unhappiness. Pain, however, even headache, is not common in acute cases of the urinary type. In severe cases headache and convulsions are not very rare, and almost invariably the latter are followed by uræmic anæsthesia. This is an interesting condition, in which there is complete blindness lasting for from an hour to several days. There are no pupal changes, and the pupils react to light, but their contraction is diminished. It is improbable that it is due to ordinar of the occipital cortex, for were it so cerebral pulses would be as common in normal children as anæsthesia, whereas they are almost unknown. Preceding a convulsion, the blood-pressure is much raised. Some hold that uræmic anæsthesia is due to a momentary spasm of the retinal arteries. Oera of uræmic origin is seen in children, but is seldom deep. Very rarely is seen a some form of uræmic resembling a tuberculous meningitis.

Vomiting in acute nephritis may be uræmic or cardiac in origin. Subcutaneous hemorrhages are not uncommon; they seem to occur much more readily in children than in adults. Anæmia is as a rule severe, but noteworthy exceptions occur. Chloemia of the gloebs may occur.

The urine is scanty, of high specific gravity, containing blood, much albumin, and an abnormally small quantity of urea. Microscopically it shows blood-cells, renal epithelium, and casts of the blood, hyaline granular, and epithelial types.

Course. In favorable cases recovery occurs in three or four weeks, but occasionally a trace of albumin may remain in the urine for several months, and yet apparently complete recovery ultimately take place.

Complications.—The complications most often met with are pneumonia, droopy of the pleural or pericardial cavities, cardiac dilatation and oedema of the lungs.

Diagnosis.—This can be made only by an examination of the urine, for the symptoms may be very misleading. Edema may be present from various causes (p. 404) without renal disease. On the other hand acute nephritis may exist with practically no constitutional symptoms at first, the child's face being well returned and showing no trace of edema.

Prognosis.—We have to consider the outlook firstly as regards life during the acute attack, and secondly as regards the ultimate condition of the kidneys.

Acute inflammation occurring in previously healthy kidneys very seldom causes the death of the patient during its acute stage. Even including those cases where the most severe symptoms are present, the majority of patients survive. The progress of the case is favourable where the urine becomes less scanty, the blood and albumin gradually diminish, and the dropsy disappears. Probably the best guide, however, is the state of the pulse- tension. Where this diminishes in the absence of cardiac dilatation, the patient may be considered to be improving.

A trace of albumin may remain in the urine for many months, and even slight recurrences of the edema be noted, with ultimate recovery. Such a course is this is, however, exceptional, and as a rule these lingering cases do not completely recover.

The prognosis with regard to the development of chronic parenchymatous nephritis is one of great difficulty. Where recovery from the acute attack has been apparently complete in three or four weeks, and the urine remains free of albumin when the child is leading a normal life, there is probably no danger of any further trouble arising. The most severe nervous symptoms, should the child survive them, do not appear to predispose especially towards incomplete recovery. Although, as has been stated, total recovery may occur after some months of albuminuria, yet such is a bad sign, and, particularly where it is associated with the persistence or reappearance of such symptoms as dropsy, headache, irritability, or with attacks of hematuria, the superintention of chronic renal disease is probable.

The ultimate outlook is much more favourable in post-scarlatinal nephritis than in the so-called primary cases.

Treatment.—The child must remain in bed, and must be kept warm by means of suitable clothing and hot-water bottles. The diet should consist of milk only. The patient should be encouraged to drink plenty of water. The most valuable remedy we have in dealing with acute nephritis is the hot-air bath. Children usually accustom themselves to it, although at first it causes a good deal of fright. During it the child may be given hot drinks. Where the skin does not act well in the bath, sweating may be encouraged by means of a small dose of pilocarpine, hypodermically (gr. $\frac{1}{16}$ to $\frac{1}{8}$) given, if preferred, with a little strychnine.

The bowels should be kept regularly open by means of saline aperients, but repeated purges are not as a rule to be ordered.

In severe cases counter-irritation to the kidneys may be beneficial. This may be carried out by means of poultices, cupping, ice-bags, blistering, or leeches. Normal saline injected high into the colon at a temperature of 104° to 108° F. is strongly recommended by Hall. A pint at least should be injected several times a day. Intravenous transfusion combined with bleeding may be ordered.

The nervous symptoms may be treated by means of rectal injections of chloral hydrate, large doses of which are well borne. Lumbar puncture will usually stop convulsions. Venesection is occasionally necessary. The heart may require symptomatic treatment by means of strychnine, digitalis, or strophanthine.

During the acute stages diuretic drugs are best withheld altogether. Should there be evidence of inherited syphilis (as in infantile cases), or of rheumatism, treatment should be by means of mercury or salicylate of soda.

Every effort must be made to obtain a total recovery. The strictest form of treatment should be carried out for at least six weeks if albumin is still present in the urine. The child should be kept in bed for not less than a fortnight after the last traces of albumin have disappeared from the urine, and the effects of all relaxations of treatment must be carefully noted. Bread and milk and bread and butter are the first foods to be added to the strict milk diet.

Where the albumin does not disappear from the urine in six weeks, the treatment must be relaxed. More food must be given. Diuretics such as diuretin may be of advantage now. Iron is also of benefit. A change to a warm climate is often most salutary.

It may seem very severe treatment to feed a child on nothing but milk for a month or six weeks, but nevertheless it appears the most rational and the most successful line to adopt. There is all the difference between a partial and a total recovery.

CHRONIC PARENCHYMATOUS NEPHRITIS.

In instances of this form of renal disease there is usually a history of a previous acute attack of inflammation of the kidneys, but not often one associated with scarlatina. In a few cases no such preceding illness can be traced.

The Symptoms may follow those of the acute attack without any intervening stage of good health, but there may be a period lasting for a few months, or even a few years, during which the patient has had no symptoms of renal disease. In most of such cases, however, although the patient has felt well, albuminuria has been persistent. Occasionally, on the other hand, recovery from the acute attack is

apparently complete, both as regards the symptoms and signs, but symptoms of chronic parenchymatous nephritis develop later.

Dropy is usually the most prominent symptom. It varies considerably in amount, fluctuating from time to time. In severe cases effusions develop into the peritoneum, pleura, and pericardium. Pallor is, as a rule, well marked, and sometimes the "large white face" is very characteristic. There are various symptoms of the chronic uræmia present, of which the most common are headache, vomiting, and diarrhoea. The urine is diminished or normal in amount, of high specific gravity, and containing albumin and hyaline, epithelial, granular and fatty casts.

The Course of the disease is an irregular one. Periods of remission and exacerbation occur. Although it may not cause death for many years, the outlook is always most unfavorable, and in many instances death occurs in three or four years. It is due to uræmia, pneumonia, or pericarditis with heart failure.

The Treatment of the disease does not differ from that adopted in the case of adults. Prophylactic treatment has been considered under the section dealing with acute nephritis.

CHRONIC INTERSTITIAL NEPHRITIS.

This is a very rare condition in children. It may be caused by inherited syphilis. It is sometimes seen in the functioning and hypertrophied parts of the kidneys, where those organs are partly functionless as a result of congenital cystic degeneration or of benign growths.

The Symptoms may be those of uræmia, or they may result from the cardiac hypertrophy and high arterial tension associated with the renal disease. Polyuria is usually present and may cause great hypertrophy of the bladder, or, from the chronic over-distention of the organ may give rise to irritation of urethra with dribbling incontinence.

As a rule the nature of the case is not suspected until the terminal uræmic symptoms appear.

MALIGNANT DISEASE OF THE KIDNEY.

A primary sarcoma of the kidney is not an uncommon condition, and is the most frequent abdominal malignant tumour in the first five years of life. It may be congenital. It occurs most frequently in children under five years of age; the second year being the commonest age. Carcinomata are very much rarer.

Symptomatology.—Swelling of the abdomen is usually the first thing noticed as being wrong with the patient. At this period of the

disease the general condition of the child as evidenced by its complexion and nutrition is often extremely good and may be very misleading. The kidney affected may grow to a great size, and cause displacement of the organs near it. The tumour is usually lobulated, and is often soft to the touch, even sometimes giving rise to a sensation of fluctuation. The colon may be felt lying over the tumour but resonance cannot always be obtained as the bowel may be flattened by the growth under it.

Hæmaturia is not a very prominent symptom. Usually the amount of blood in the urine is small, and often can only be recognized by the microscope. Occasionally the bleeding is profuse. There is seldom much actual pain in the region of the tumour, but as the result of the large size of the growth vomiting or dyspepsia may be produced by pressure on the stomach and lungs. As the tumour continues to grow, the child wastes and becomes cachectic, but this is not as a rule noticeable until late in the disease.

The secondary deposits which occur in the liver, lungs, retro-peritoneal glands and elsewhere, rarely give rise to any definite symptoms.

Diagnosis.—The recognition of the disease has usually to be made by palpation of the abdomen. Hæmaturia is a sign of great positive value, but so often is this absent that a negative result of the examination of the urine is no aid in diagnosis. The rapid growth of the tumour and the consideration of the age of the child as a rule render the diagnosis clear. The tumour may be mistaken for a hydrosphrotic kidney, an enlarged spleen, an ovarian cyst, or retro-peritoneal tumour.

Prognosis.—The outlook is extremely serious. Although with early operative treatment some instances of permanent recovery have been reported, they are very exceptional. Recurrence within two years of the operation is the rule. If not removed, the growth usually causes death in about six months.

TUBERCULOSIS OF THE KIDNEY.

This has been dealt with on p. 143.

HYDRONEPHROSIS.

We may consider this under the headings of congenital and acquired hydro-nephrosis.

Congenital Hydro-nephrosis.—The condition is usually bilateral. Often it gives rise to no symptoms during life, and is discovered only after the death of the patient, which usually takes place within a year of birth. Occasionally the abdomen becomes enlarged as the result of the hydro-nephrosis. Urinary symptoms are seldom present unless

a secondary septic infection occur. The kidney tissue may undergo pyo-nephroses and interstitial changes. In some cases the kidneys, although not much enlarged, contain only a small amount of renal tissue; the ureters are enormously wide and thickened, resembling small intestine, and the bladder much hypertrophied. Evidence of obstruction of the urethra may be found, but is not seldom entirely wanting. Death is usually due to broncho-pneumonia and wasting, and not directly attributable to the condition of the kidneys.

Unilateral cases may give rise to no trouble until the child is a few years old, when the swelling of the kidney may cause a large abdominal tumour. Where certainly unilateral the treatment is preferably by nephrectomy.

Acquired Hydronephrosis.—It is not uncommon to find some dilatation of the pelvis of the kidney in newborn children with phthisis. In older children hydronephrosis may be due to renal calculus, to an abdominal tumour pressing upon the ureter, to mobility of the kidney or to polypus (p. 302).

Traumatic hydronephrosis is a peculiar condition which may arise within a few weeks of some severe injury to the kidney. It does not produce any constitutional symptoms, and may disappear spontaneously. Should it progress to the formation of a large tumour, it may require surgical treatment.

RENAL CALCULUS.

In young infants, uric acid calculi of small size are very commonly found post mortem, but they seldom give rise to any symptoms during life. Occasionally, however, they cause slight hæmaturia and attacks of abdominal pain simulating intestinal colic, but recognizable as the subsequent passage of uric acid in the urine.

In older children, severe attacks of renal colic are sometimes seen. Often, however, the symptoms produced by a stone in the kidney are much less definite, and consist of some frequency of micturition and the occurrence of pyuria. Hydronephrosis may occur as the result of impaction of the stone in the ureter. The Roentgen rays may be of great value in the detection of a calculus, but often the diagnosis is one of very great difficulty.

Treatment.—For most cases in older children operative measures are necessary.

URIC ACID INFARCTION.

In autopsies upon children under six months of age, it is very common to find deposits of uric acid at the apices of the renal pyramids and radiating from these towards the periphery of the organs. These

are spoken of as *lime acid* infarcts. They occur in the new-born and also in young infants who, from the presence of vomiting or diarrhoea, have passed less than the normal amount of urine. They have also been found in the kidneys of still-born children. Associated with this induration, small granules of lime acid are often found lying loose in the pelvis of the kidney. As a rule, their presence is not recognizable during life, but such symptoms as may be produced are probably referable to the small calculi.

ACUTE PYELITIS.

In most instances pyelitis is secondary to cystitis due to the *B. coli* syndrome, and as such may be considered under cystitis (p. 459). It may also arise in connection with renal calculi, renal tubercles, or associated with some of the acute fevers, such as scarlatina or typhus. It may, if severe, set up pyelonephritis and pyonephrosis.

PRIMARY PYELITIS.

Etiology.—An extremely interesting condition is met with in certain cases in which there is little or no evidence of cystitis at all events at the beginning of the illness. Such cases give rise to most alarming and perplexing symptoms*. They are due to the *B. coli* syndrome, and occur much more commonly in girls (84 per cent.) than in boys. The patient is usually under two years of age, and the disease most frequently arises between the third and ninth months. Many cases arise in bottle-fed children than in those fed on the breast. No seasonal or hygienic influences can be traced.

Symptomatology.—The chief point of interest in these cases lies in the fact that the constitutional symptoms are remarkably severe while the local symptoms are very slight, and often at first entirely absent. Owing to this there is great difficulty in recognizing the disease.

The onset of the general symptoms is usually sudden. The temperature runs up to a great height, the child becomes moribund and convulsed. The respiration-rate is much increased. Delirium, delirium, vomiting and squinting may be present. A remarkable symptom is seen in this disease, namely, the presence of rigors. This is practically the only disease which causes rigors in infancy. Another symptom which is commonly seen is that of fainting attacks, in which the child becomes extremely pale and collapsed. These paroxysmal faints may be the first symptoms of illness. There is a leucocytosis of from 15,000 to 25,000. In older children there may be pain and tenderness in the region of the kidneys.

* John Thomson, *Quart. Jour. of Med.*, April 1922.

The course of the disease is very irregular. The temperature conforms to no set type. There are hours, even perhaps days, when the child seems comparatively well, and will sit up playing with its toys, although the temperature may be raised. The respiration-rate, although much quickened, causes little discomfort. But at irregular intervals there reappear the alarming symptoms, the temperature reaching 104° to 105° , and rigors, convulsions, and faint-fits being present. The temperature may show extremely large and rapid oscillations.

The focal symptoms are at first often entirely absent. Increased frequency of micturition is the most constant early sign. The urine is highly acid, and contains the *B. coli*, but no pyuria may be present.

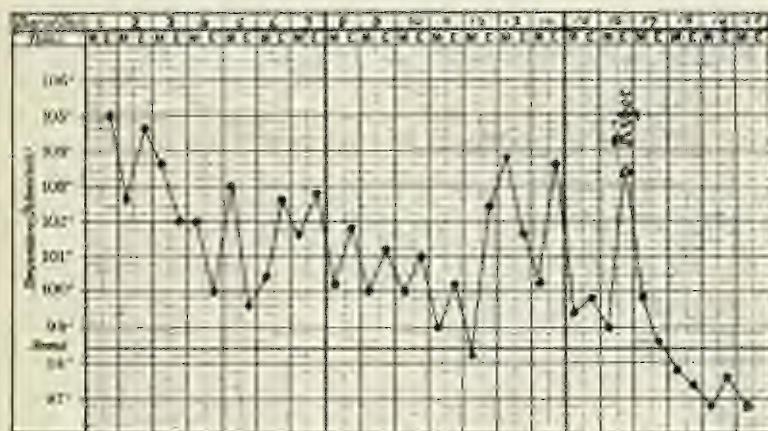


Fig. 26.—TEMPERATURE COURSE: Primary Pyklitis in a female infant of six months (Lindqvist).

for the first few days. Later, often as the general symptoms begin to become less severe, the urine contains pus, and symptoms referable to cystitis (p. 405) may develop.

Diagnosis.—As will be seen by the description of the disease, there is little to suggest its nature unless its possibility be borne in mind. In cases of unexplained pyrexia in children, the examination of the urine is never to be omitted. In the early stages there is bacteriuria, while after a few days pyuria occurs. The presence of rigors, fainting-attacks, and very great oscillations in the temperature are suggestive clinical points. Lastly, the diagnosis may be reached by the exclusion of other possibilities.

The increase of the respiration-rate may well give rise to a suspicion of primary pneumonia. Certain phases of the disease strongly resemble

the picture of acute meningitis. Irregularities in the fever, intermissions in the symptoms, and the occurrence of faint turns and rigors point towards acute pyelitis.

Prognosis.—Recovery occurs in many of the cases in which the most alarming symptoms are present. The difficulty in diagnosis adds to the danger of the disease. Death occurs in a minority of the cases, and is due to infection of the substance of the kidneys.

Treatment.—The child should be encouraged to drink plenty of fluid. The bowels should be opened by calomel, and a daily aperient of sodium phosphate, which tends to turn the urine alkaline, may be given. Every effort should be made to render the urine alkaline in those cases which are due to a pyre infection by *B. coli*. For this large doses of sodium citrate may be employed, from 50 to 150 grains being given daily. Ureteropneum may also be given, even up to 10 grains four-hourly for a child of two years. For the mixed infections of older children, Dr. John Thomson advises against the use of alkalies. In cases threatening to become chronic a vaccine prepared from the patient's organism should be administered.

ŒDEMA.

Œdema occurs in new-born infants (œdema neonatorum) associated with sepsis and a subnormal temperature. The dorsal surface of the hands and feet and the face are the most common sites for the œdema. Treatment should consist of warmth and careful dieting.

Some rare cases are classed as "congenital œdema." The œdema, which is of the ordinary type, is present at birth and persists. The feet are most commonly affected. Still rarer is the condition known as "hereditary œdema of the lower limbs." This disease, which is both hereditary and familial, is usually but not constantly congenital. One or both legs may be affected. The condition is persistent but harmless except for the inconvenience it causes. The enlargement of the affected parts may become very marked, so that the condition is sometimes termed "chronic congenital elephantiasis." No treatment is satisfactory. Bandages, massage, rest and tonics may be tried.

It may be of use here to consider two types of œdema which are not of renal origin, as they not seldom give rise to mistakes in diagnosis.

Œdema with Diarrhea.—General œdema in children may occur in cases of diarrhoea of a severe type without acute nephritis. It is most commonly seen in the acute infective diarrhoea of infants, but also occurs in older children, as for instance, in cases of tuberculous enteritis. In the former group it is usually but not invariably a sign of fatal significance. It is not uncommonly associated with urticaria or a purpuric rash on the lower part of the abdomen.

Œdema of the Eyelids.—This, when combined with pallor of the

face may give rise to a suspicion of nephritis. In the absence of renal disease, and excluding such cases as are associated with diarrhoea, oedema of the eyelids is a very characteristic sign of pericarditis. Both the rheumatic and the tuberculous cases show it, but it is not seen in psalient pericarditis. The upper lids are usually more swollen than the lower. Puffiness round the eyes is seen in cases of enlarged bronchial glands, whooping-cough, and measles. In the last-named it is accompanied by conjunctivitis.

III.—DISEASES OF THE BLADDER.

CYSTITIS.

The *B. coli* accounts for the great majority of cases of cystitis in children. The condition is a common one in infants and young children, and is probably much more frequently present than is as yet recognized. Girls are more often affected than boys. The infection may be through the urethra, by the lymphatics from the intestine, or through the circulation.

Predisposing causes to cystitis are prolonged constipation, chill, vulvovaginitis, thread-worms, and diabetes.

Occasionally cystitis is secondary to a diphtheritic infection of the vulva. Various other bacilli and cocci, including the pneumococcus, have been reported as causing the condition.

Chronic cystitis may arise from an acute attack, or may be associated with vesical calculus. Tuberculous cystitis is not a common condition in children.

Symptomatology.—"Colocystitis" is seen in varying grades of severity. It frequently exists without any symptoms, as is suggested by the length of time by which pyuria may collect all symptoms during convalescence from a recognized attack. Certainly local symptoms are often entirely absent, and the condition only diagnosed by a routine examination of the urine.

In mild cases it is most commonly for incontinence of urine that the child comes under observation. This, which may be either nocturnal only, or nocturnal and diurnal, is associated with some frequency of micturition. The constitutional symptoms in this type of case will probably be limited to some pallor, with a rise of temperature at night.

In more severe cases, in addition to the incontinence and increased frequency of micturition, there may be dysuria and tenderness in the region of the bladder. The child has a toxic appearance, is pale and ill-looking, and the temperature is permanently raised, although tending to be rather remittent in type. In this condition the mistaken diagnosis of typhoid fever may easily be made unless the urine be examined.

In cases of still greater severity, the pelvis of the kidneys become affected (pyelo-cystitis), and the series of symptoms already given under pyelitis (p. 402) become more or less in evidence.

The urine in calycystitis is acid in reaction and contains a variable quantity of pus. *B. coli* is always present.

Diagnosis.—There is no little danger of overlooking or misinterpreting the symptoms in cystitis. The diagnosis is to be made only by an examination of the urine. This should be done as a routine, but especially in cases of incontinence and of unexplained pyuria in children.

Prognosis.—The slight cases recover rapidly. In more severe attacks recovery is delayed considerably; but as a rule the symptoms quickly abate, although the pyuria may continue for many weeks after. Where there is evidence of pyelitis, the danger to life is increased.

Treatment.—Where the temperature is raised, even though it be at night only, the patient should be kept in bed. The child should be allowed plenty of fluid to drink. For drugs, large doses of alkalis, sodium bicarbonate or sodium citrate, may be given in order to render the urine alkaline. Urotropine is here of great value as a disinfectant, appearing to act much more efficiently when the bladder is involved than where the pelvis of the kidneys are bearing the brunt of the attack. 3-grain doses may be given three daily to a child of three or four years. The urotropine and alkalis may be prescribed together in a mixture. In severe cases the bladder may be washed out.

When the symptoms have all passed off, but the bacilluria and pyuria continue, improvement is sometimes brought about by allowing the child to get up. In retarded cases a vaccine prepared from the patient's own organism may be of benefit.

VESICAL CALCULUS.

The diagnosis of stone in the bladder frequently falls upon the physician. It is not an uncommon condition in children, and produces increased frequency of micturition with pain during or after the act. The flow of urine may be suddenly stopped. Incontinence is common, while hematuria and rectal prolapse are not very infrequent. The urine contains mucus and pus, although sometimes in small quantities only. Hematuria is a much less constant symptom.

The symptoms of vesical calculus may be simulated by an adherent peapate, thread-worms, and cystitis without stone. Where the presence of a calculus may be reasonably suspected, a skintagm of the pelvic region should be taken. With children, this is preferable to the passage of a sound.

ENURESIS.

The subject of incontinence of urine is discussed here under diseases of the bladder, in order to emphasize the fact that it is not necessarily due to a nervous disorder, but may arise from some local condition. Enuresis is a very common complaint in children, and deserves much more careful consideration than is sometimes given to it. Often the success of treatment depends solely upon the amount of care which has been taken in investigating the case. Enuresis is a symptom and not a disease, and no one remedy can cure such and every case.

Etiology.—Incontinence of urine may of course be due to some serious nervous disease such as idiocy, cerebral tumour, or myelitis; or it may be dependent upon some gross malformation of the urinary tract, whereby retention of urine in the bladder is rendered impossible. With such conditions we are not concerned, for we have to consider here the ordinary cases in which incontinence occurs without any very obvious condition to account for it.

In investigating such a case, we have to inquire into the following points: Has the enuresis existed from birth, or is it an acquired condition? Does it occur at night only, or is it also diurnal? Is it associated with incontinence of feces? Is there increased frequency of micturition? We have further to examine the urine, particularly in acquired cases.

Where the tendency to enuresis has existed since birth, and the child has never obtained full control over his bladder, the nervous constitution of the patient and imperfect training are likely to be of more importance in the causation of the habit than any local or reflex conditions. Such may, however, keep up incontinence in a neurotic child. Acquired enuresis on the other hand, may be due either to some condition which directly or reflexly causes nervous instability, or to some local disease of the genito-urinary tract.

If diurnal as well as nocturnal enuresis, in the absence of mental deficiency, is likely to be due to some severe local irritation or inflammation, such as cystitis or vesical calculus.

Rectal incontinence is seldom seen associated with enuresis apart from mental insufficiency.

Increased frequency of micturition, where it is well-marked, and especially if accompanied by distress, points strongly to some local cause for the condition. It must be remembered, however, that where enuresis, congenital or acquired, is due to nervous instability, some increase in the frequency of micturition is likely to be present, for such children cannot contain their urine for a normal length of time.

We may therefore consider the causes of enuresis under the headings of nervous, reflex, and local.

Nervous Causes.—In congenital cases an inherited neurotic taint is the commonest cause of enuresis. Acting in association with this

and improper training of the child and possibly some reflex or local condition. Sometimes enuresis occurs in several members of a family, while occasionally it may be possible to get a history of one of the parents being similarly afflicted in childhood.

Nervous instability may, however, be acquired, or perhaps more frequently increased by some acquired condition, and this be the cause of enuresis starting in a child previously clean in its habits. Thus enuresis may begin during convalescence from illness or during a period of anaemia. Nocturnal incontinence of urine is a common symptom in rheumatic children who are the subjects of what I have described elsewhere as latent chorea.

Amongst the nervous causes of incontinence must be mentioned mental deficiency and deterioration, epilepsy and gross nervous lesions. I have known a case of juvenile tabes to come under observation for bed-wetting. This is of course one of the rarest causes.

Reflex Causes.—Enlarged tonsils and adenoid vegetations frequently cause nocturnal enuresis, acting by the production of partial asphyxia during sleep. In many of these cases the enuresis is accompanied by night-terrors. Other causes of disturbed sleep, such as constipation, may act in the same way. The presence of thread-worms, by producing much local irritation at night, may induce nocturnal incontinence of urine.

Local Causes. Many affections of the genito-urinary tract may cause the onset of enuresis or may prevent habits of cleanliness being acquired by nervous children. Of these the most common are bacilluria, cystitis, vesical calculus, hyperacidity of the urine, adherent prepuce, valvovaginitis, and thread-worms. A contracted bladder is a rare local cause.

Prognosis.—Acquired enuresis, as a rule, rapidly improves under appropriate treatment, except where due to some permanent organic disease of the nervous system. In the cases which have existed since birth and which are dependent upon the patient's nervous instability, the outlook is less favourable. With care, however, these cases can usually be cured, while failing recovery under treatment, the enuresis generally ceases at the age of puberty.

Treatment.—Where due to some local or reflex cause, enuresis must be treated by the removal of the morbid condition from which it arises. There is no need for the repetition here of the treatment to be adopted in such instances, as it has been dealt with in various parts of this work.

Enuresis due to an acquired nervous instability, recent illness, anaemia, or latent chorea, may be treated by tonic measures or by such drugs as are necessary to restore the patient to health. The climatic cases are often benefited by a mixture of sodium-sulphate and sodium bicarbonate, followed later by tincture.

We are here chiefly concerned with the treatment of those cases which have existed since birth, and which appear to be entirely due to the neurotic condition of the patient.

A few points in the general treatment may be mentioned first. The child's constitution should be built up as well as is possible. While particular care must be taken not to overtax the nervous system, fresh air and physical exercise are of value by improving the appetite and nutrition, and by ensuring quiet sleep at night. The diet should be plain and wholesome. Food should not be given later than at tea-time, while tea and coffee are best prohibited. It is generally very unwise to send the patient to a boarding-school, as his unfortunate habit is usually regarded by his schoolfellows as due to criminal negligence on his part, and receives punishment accordingly.

Of drugs, belladonna is of most value. It must, however, be administered in doses as large as can be borne without discomfort. Seven or ten minims of the tincture may be given at first, and the dose rapidly increased. Bromide or chloral is often given with the belladonna, but these sedative drugs seem to have little good effect. Strychnine is of more value, and a combination of belladonna and *nux vomica* is perhaps the most powerful of any in the cure of enuresis. Many other drugs are recommended, but rarely succeed where belladonna and strychnine have failed. Thyroid extract does definite harm in most cases; but in a few, particularly in fat, lethargic, but in no way mentally-deficient children, small doses of this preparation act wonderfully well in relieving the symptom of enuresis.

Of the position of drugs in the treatment of enuresis, we may say that, while many of them do good for a time, a permanent cure very seldom results from their use alone.

Most important of all in the treatment of nocturnal enuresis is the question of training the child to overcome the habit. During the daytime the patient should be encouraged to micturate as seldom as possible, in order to accustom the bladder to retain successfully normal quantities of urine. At night the child should be awakened every two hours at first, and given the opportunity of passing urine. After a week the intervals may be lengthened to three hours, and later to four hours.

Often it is best to have the child removed from home, and in obstinate cases this is almost essential. The admission to a hospital ward is very frequently quite sufficient to stop the habit at once, but should incontinence recur, the routine sketched above should be carried out. The child should be made to understand that there is no necessity for the habit, and that he will be expected to keep his bed clean, and be given every opportunity to do so. Corporal punishment is never to be inflicted for incontinence, but rewards for each night in which the bed has not been wetted are frequently valuable aids to treatment.

Such a plan as this, removal from home and strict training, very rarely fails to arrest the habit; but, unfortunately, only too often after

If the patient has been home for a week or two the incontinence disappears. It is necessary, therefore, to instruct the parents that the responsibility of the cure lies entirely in their hands.

IV—DISEASES OF THE GENITAL ORGANS.

The malformations and diseases of the genital organs of both sexes belong as a rule to the province of surgery rather than to that of medicine.

Vulvovaginitis.—The severer forms of this condition are due to the gonococcus. Others, which are less serious, less intractable, and less infectious, are due to streptococci. Lack of cleanliness and the presence of thread-worms predispose to such an infection.

The reader is referred to the description of the gonococcal cases on page 207.

Hæma Pudendi.—This is a rare condition which is similar, except for its localization, to carcinoma. It is, however, less common. It occurs in wasted female children, following the infectious fevers, notably measles. There develops a bruise swelling of the labia majora, in which gangrenous changes quickly make their appearance and, spreading, lead to much destruction of tissue.

The treatment of hæma pudendi consists of excision or cauterization.

SECTION IX.

DISEASES OF THE SPLEEN, BLOOD, AND GLANDS.

I.—DISEASES OF THE SPLEEN.

Normally the spleen in birth weighs about $\frac{1}{2}$ oz., and at the end of the first, second, and third years, $\frac{3}{4}$, 1½, and 1¾ oz. respectively (Hoffa). Owing to the firm state of the abdominal wall in children the organ is palpated much more easily than in adults, and may often be felt lying in its normal position just under the costal margin. The examiner should stand on the patient's right side, and pass his right hand, which has been warmed, over the abdomen, reaching the left costal margin at the axillary line. If enlarged, the spleen will be felt as a jutting space the radial border of the index finger. Care must, however, be taken to carry the examining hand well back into the loins, lest it should pass upwards in front of the spleen. In children who will not lie quietly, the examination may be made with the child in a sitting posture, the body being bent well forward to relax the abdominal muscles.

Displacement of the spleen in a downward direction may occur where the left side of the thorax is small, as in rickets, or may be produced by a left-sided pleural effusion, or by a much enlarged liver.

Enlargement of the spleen is of common occurrence in children, and is often of considerable importance in the diagnosis of disease.

In infants a palpable spleen is most commonly associated with rickets in which there are both displacement and enlargement of the organ. At the same age or later, inherited syphilis is a less common cause, but usually produces greater splenic enlargement than rickets.

Many acute infections cause an increase in size of the spleen, and of these the most important are pneumococcal infections, tuberculosis, and typhoid, while any of the acute specific fevers of childhood may exceptionally cause some splenic enlargement. It is very necessary to bear in mind that in pneumococcal infections, especially when at all prolonged, enlargement of the spleen is very commonly present. A difficulty may arise in this way—a case of pulmonary disease, at first regarded as an ordinary instance of pneumonia, does not get well, the high fever and the dyspnoea remain unabated at the end of a week or ten days, and the question arises, Is it a tuberculous process? The spleen is found to be definitely but moderately enlarged, and on the

sign a diagnosis of tuberculosis is often erroneously made. It must therefore be remembered that although in such a case a moderate enlargement of the spleen is somewhat in favour of tuberculosis, it is very far from being definite evidence of that disease.

In the differential diagnosis of general tuberculosis and typhoid (p. 20) mistakes sometimes arise on account of the condition of the spleen. A hard spleen is said to be in favour of tuberculosis, and while this is the case, it must be emphasized that in typhoid the spleen occasionally feels extremely hard. Doubtless post mortem the spleen is found to be soft, but during life, where there is great congestion of the organ, its capsule is tightly stretched and so gives the impression of hardness to the examining hand. In these conditions any great enlargement of the spleen suggests that there has been chronic tuberculosis, to which is now added an acute terminal generalized infection. In tuberculosis the spleen is nearly always enlarged in response to the toxæmia present, and not as a result of local tuberculous deposits.

The spleen may be enlarged in other infections, notably in cases of malignant endocarditis. In rheumatic heart disease the spleen is very seldom palpable, and any definite enlargement of it should suggest at once that the valvulitis is of the malignant type.

In certain blood-conditions increase in the size of the spleen is present. In anaemia due to rickets, syphilis, tuberculosis, chronic parasitæmic infections (e.g. a latent ringworm), and in some chronic forms of septicæmia and toxæmia, the spleen may be enlarged. Such is also the case, usually to a greater degree, in acute lymphatic leukaemia, splenic anaemia of infants (Von Jaksch), and Hodgkin's disease.

Less common causes of splenic enlargement are amyloid disease, malaria, hydatid disease of the liver, family xanthomic jaundice, Hæser's cirrhosis and primary splenomegaly.

PRIMARY SPLENOMEGALY.

(*Splenic Anaemia*).

Etiology.—Very occasionally there arises in older children a condition simulating precisely that known in adults as primary splenomegaly or splenic anaemia. The disease can be closely copied by the results of tuberculosis or syphilis, but is a separate entity from either; nor does there appear any true relationship between primary splenomegaly and those cases classed as Von Jaksch's anaemia (splenic anaemia of infancy).

The disease is here classed under diseases of the spleen in accordance with the view that it is due to endothelial proliferation within that organ (Holliston). The evidence afforded by the results of splenectomy appears to warrant its inclusion in this group. Dr. G. A. Sutherland regards the disease as due to a loss of vasomotor control of the splenic vessels, comparable to the condition of the thyroid vessels in Graves' disease.

As it arises during childhood it is seen chiefly in older children of six years old and upwards. It may co-exist in more than one member of a family.

Symptomatology.—In the first stage of the disease enlargement of the spleen is probably present before any constitutional symptoms are noticed.

In the second stage, and this is when the patient usually comes first under medical supervision, progressive anemia develops and reaches a severe grade. The spleen is now much enlarged, often down to or even below, the level of the umbilicus. The patient becomes intensely pale and suffers from the usual symptoms of severe anemia. In children the lemon-yellow coloration, such as is seen in adults with pernicious anemia, may be noted. This suggests that an excessive destruction of red blood cells is proceeding. There is no enlargement of lymphatic glands.

The blood shows a great diminution in the number of erythrocytes and in the percentage of hæmoglobin. Normoblasts are numerous. The white cells are diminished in number (leucopenia) and show a relative increase of lymphocytes.

As in other severe anemias irregular pyrexia and hæmorrhages may be present. The occurrence of repeated hæmatemesis, rather a feature of the disease in adults, does not seem to be common in children. It does not necessarily depend upon cirrhotic changes in the liver. Hæmorrhages anywhere else may occur and tend to become common towards the latter part of the second stage of the disease.

In the third stage there is a progressive increase in the symptoms mentioned with the development of hepatic cirrhosis (splenomegalic cirrhosis). This terminal stage is often called "Banti's disease." The liver is palpable a short distance below the costal margin and feels hard and, at a late stage, possibly hob-nailed. Ascites is uncommon, jaundice still rarer. Hæmorrhages are common at this stage and a patchy pigmentation of the skin may develop.

Morbid Anatomy.—In an autopsy on a boy aged eleven years who had died in the last stage of the disease, Dr. Hale White found the liver to appear exactly like a hob-nailed atrophic cirrhotic liver. It weighed only 26 oz. There was much cirrhosis, chiefly intralobular but considerably multilobular; very little intracellular. The spleen showed much hyperplasia of the splenic pulp and considerable endothelial proliferation in the blood sinuses.

Diagnosis.—The exclusion of tuberculosis or syphilis as the cause of anemia with enlarged spleen must be carefully carried out. Where this is possible the great reduction in the red cells, hæmoglobin and leucocytes and the very marked enlargement of the spleen are eminently suggestive of primary splenomegaly. Similarly the lemon-yellow tint

III. DISEASES OF SPLEEN, BLOOD, AND GLANDS

of the skin may be very characteristic. Both pernicious anemia and spleno-medullary leukemia are practically unknown in childhood. In Von Jaksch's anemia the blood-picture may be similar to that of primary splenomegaly, but the former disease arises in infancy, may show glandular enlargement, is often related to syphilis and rickets, may show a leucocytosis and tends to get well under treatment.

In the third stage of the disease a past history of splenic anemia is of vast importance. Failing this, we have to differentiate "Banti's disease" from Harrot's cirrhosis and from ordinary atrophic cirrhosis of the liver. In Harrot's cirrhosis the spleen is also very much enlarged, but here the liver is very big and is usually smooth, while there is a history of jaundice persisting over a long period. From atrophic hepatic cirrhosis of the type usually but not always due to alcohol, Banti's disease may be differentiated by the very great enlargement of the spleen, and the rarity of ascites and jaundice. For the accurate diagnosis of Banti's disease, however, a past history of splenic anemia is almost essential.

Course and Prognosis.—Although periods of improvement may occur the natural course of the disease is towards a fatal ending. In adults it is said that death may not occur for eight or ten years, but in children the disease is more acute and probably it would be correct to limit its course to three or four years of the most. With the onset of hepatic cirrhosis life is not likely to be prolonged beyond a year.

Treatment.—Hygienic measures and drugs are of little use. Temporary improvement may occur under arsenic.

Excision of the spleen appears the only measure of value, and with it the results are extremely hopeful. In dealing with early and suitable cases in children the death-rate from the operation is probably extremely small. Obviously where the tendency to hemorrhage is well-marked the risk of the operation is much increased. So far as at present appears splenectomy seems to hold out a promise of a lasting cure. Certain it is that the immediate improvement in the blood-picture and general condition of the patient which follows the operation is extremely striking. Such a well-marked and immediate turn for the better as occurred after splenectomy in two cases under the care of my colleague, Dr. Sutherland, seems strongly to suggest that this is the correct line of treatment in such early cases in which it is practicable (*Lancet*, Dec. 24, 1909).

II.—DISEASES OF THE BLOOD.

Normally in infants and children the blood shows certain differences from that of adult life. For the most part the blood of the child is

more dilute than that of the grown-up. The chief exception to this rule is seen in the new-born. At birth the red cells are in excess, about 51 to 5 millions, and the percentage of hæmoglobin in the blood is about 110. At this time, also, a few normoblasts are usually present. By the sixth month, however, the red cells number between 4 and 5 millions, and the hæmoglobin is reduced to about 70 per cent. The normoblasts disappear, except in premature infants, in a few days. As the child grows the number of red cells remains the same, but the hæmoglobin begins to rise slowly after the second year, until in puberty the blood is of the adult type.

The white cells are of the same kinds as in later life, but differ in their relative and absolute numbers. At birth they number about 15,000 per cmm., and at the end of the first year about 14,000. They gradually decline to the normal adult figure as the child grows. In infancy and early childhood there is a relative increase in the number of lymphocytes present in the blood, about 40 per cent of the white cells being of this variety.

CHLOROTIC ANÆMIAS.

Chlorosis.—The disease of young women, known as chlorosis, is not common in children. It is, however, occasionally seen in girls of eleven or twelve who suffer from constipation. In such cases the shortness of breath, the headache, and cramps in the legs are seen, but the greenish tint of the skin is very rarely observable until later in life. The heart shows the usual hæmic brain, and possibly slight dilatation and irregularity.

Secondary Anæmia.—Anæmia of the chlorotic type—that is to say, with some diminution of the number of red cells and much lowering of the percentage of hæmoglobin—as in both infants and children attributable in most cases to some recognizable disorder or disease. It is important, then, to bear in mind that the blood-picture of chlorosis as seen in children is usually to be regarded as evidence of a secondary anæmia, so that in each case search may be made for the cause of the condition.

In infants such anæmia may be due to a large variety of causes. Of these, the most common are: malnutrition due to improper feeding or imperfect assimilation; chronic gastro-intestinal indigestion, rickets, tuberculosis, and inherited syphilis; and it is to be noted that in each of the three last-mentioned diseases there is likely to be enlargement of the spleen.

In older children, with the exception of rickets, the same causes may be productive of this type of anæmia, but in addition other infections must be noted. Of these the most important are rheumatism and diphtheria, both of which produce a profound anæmia. In particular, rheumatic anæmia requires emphasis, as in London it is one of

the commonest types seen in children after the seventh year. It is therefore of great importance to enquire into the matter of sore throats and growing-pains, to examine the child for signs of dilatation of the heart, and to look for the earlier manifestations of chorea, such as fidgetiness, headache, untidiness, maturation, or such evidences of acquired nervous instability as night terrors, somnambulism, habit-spasms, and the like (p. 155). It is not of course to be supposed that all anæmic children who are nervous and have dilated hearts are the subjects of rheumatism, but a very large number of them certainly do come under this category. The importance of the recognition of this fact is twofold: first, the earlier we learn that a child is anæmic, the greater is the chance of preventing serious heart disease; and second, with the presence of active rheumatism, as evidenced by recent toxicæmia, muscular pains, or a persistent slight rise of the nocturnal temperature, the anæmia is not likely to yield to treatment by iron until after a course of salicylate of soda.

Chol sepsis requires attention in the consideration of anæmia in other children.

In the secondary anæmia of a severe grade the red cells become considerably diminished in number and show poikilocytosis, while there develops some increase in the number of the lymphocytes.

VON JAKSCH'S ANÆMIA.

Etiology.—This condition is known by the names *splenic anæmia of infancy* and *pseudo-leukæmia infantum*; but as it has nothing to do with the splenic anæmia (primary splenomegaly) of adults or with pseudo-leukæmia (Hodgkin's disease), it is preferable to name the condition after Von Jaksch, who described it in 1889. It is doubtful whether it is a disease sui generis, or simply a secondary anæmia of great severity.

As originally described it is a disease of infancy (6 months to 2 years) and is in most cases associated with rickets, and in some with syphilis. Many cases in twins have been reported.

Symptomatology.—The patient is extremely pale, but is often well nourished. The abdomen is large, because of the greatly increased size of the spleen. Constipation is usually present.

The spleen is considerably enlarged, and is very firm; in some cases the liver is also increased in size. In about half the cases the lymphatic glands of the neck, axillæ, or groins are moderately enlarged. The blood shows that the red corpuscles are much reduced in number (perhaps to 2,000,000 per cmm.), and poikilocytosis is marked. Nucleated red cells are present: usually normoblasts, but in the worst cases megakoblasts are found. The hæmoglobin is greatly diminished in amount, and may reach as low a figure as 15 per cent. With these changes is usually seen an increase in the number of white cells present.

in the blood (from 10,000 to 50,000) of which as a rule the lymphocytes show the largest increase in number. In some instances, however, with a relative increase of lymphocytes, the absolute number of the white cells is diminished (leucopenia). Myelocytes may be present.

Diagnosis.—The age-incidence of the disease differentiates it from Hodgkin's disease and primary splenomegaly.

From a severe secondary anemia due to rickets, or inherited syphilis, or tuberculosis, the diagnosis can hardly be insisted upon, as many authorities hold that the cases are identical. Great enlargement of the spleen, with the presence of a leucocytosis and absence of the signs of syphilis and tuberculosis, would tend to put the case into the group of Von Jaksch's anemia.

Prognosis.—The outlook as regards the anemia is good, although the disease tends to be chronic in its course. The enlargement of the spleen may remain after the patient has recovered. The prognosis should, however, be guarded, as there is danger of death from some intercurrent disease, notably broncho-pneumonia. Probably about 75 per cent recover entirely (Custley).

Treatment of Anemia.—In the treatment of anemia of the chlorotic type in children, our first duty is to examine for any possible cause of the condition. The prevalence of secondary anemia has been already insisted upon, and it is only necessary to mention here that gastro-intestinal indigestion, rickets, syphilis, rheumatism, or any other cause of anemia, should be treated where present.

Apart from this consideration, the treatment follows the lines adopted in adults. Fresh air, sunshine, and good food are of prime importance. Iron may be given in the diet in the form of the yolk of egg, raw meat juice, or green vegetables. For older children the perchloride of iron is the most useful preparation. Younger children may better be given the syrup or the compound syrup (B.P.C.) of the phosphate of iron in doses of $\frac{1}{2}$ –2 dr. These may be prescribed with cod-liver oil if desired (p. 74). "Ferrolcum" is a useful preparation of iron and cod-liver oil. In combination with iron arsenic is sometimes of service, while it is the only drug of value in leukemia and Hodgkin's disease.

ACUTE LYMPHATIC LEUKÆMIA.

This is a disease of no little interest, and is not very uncommon among children. In most cases the patient is under four years of age. Both sexes are equally affected. As a rule the first symptoms are those of increasing pallor and weakness, which may develop with extreme rapidity. Hemorrhages into the skin, gums, and retina, or from the bowel or kidneys, are present in a large proportion of the cases, and may be very early in their appearance. The child is not

uncommonly brought to the doctor because of the sudden onset of a purpuric rash. The spleen is usually enlarged, often as far as the level of the umbilicus, and the liver is also frequently increased in size. The glands in the neck are enlarged in the majority of cases, while in some there is a general enlargement of the superficial glands of the body. At first, these glands may be normal in size. The temperature is high, often irregular, but may fall to normal during a period of improvement in the disease. Vomiting is common, and may be an early feature. Hematemesis is rare. Diarrhoea may occur, and blood may be passed by the bowel. An interesting point is the enlargement of the kidneys, which may be sufficiently marked to be recognizable by palpation of the abdomen. These organs post mortem are found to contain areas of hemorrhage and to be greatly congested with lymphocytes. Hematuria is rare, but albuminuria is present in about half the cases. The blood shows a great increase of the white cells present, the majority of which are lymphocytes. It is to be noted, however, that during the last week or two of life the number of the white cells is much reduced and may soon become normal, but the relative increase of the lymphocytes remains. The red corpuscles are diminished in number.

The origin of the disease is very obscure. In children the extreme rapidity of its onset and course, the death may result in a week, suggests a bacterial infection.

A valuable paper on this disease by Forbes and Langmead may be found in the *Proceedings of the Royal Medical Society* for May, 1908.

Prognosis.—The course of the disease in children averages five or six weeks (one week to seven months), and, although occasionally short remissions may occur, the disease is always fatal.

Treatment by means of arsenic or stroyl should be tried.

CHLOROMA

is an extremely rare condition, which shows a blood-picture similar to that of acute lymphatic leukaemia, but in addition sarcomatous growths occur in the skull-bones. Post mortem these are found to be of a greenish colour. It is possibly a connecting link between acute lymphatic leukaemia and lymphosarcoma.

SPLENO-MEDULLARY LEUKÆMIA.

This is a disease of very great rarity in childhood, only about a dozen cases being as yet reported.

PURPURA.

This is considered most conveniently under two headings: Acute

symptomatic purpura, cases in which we can trace some cause for the condition; and second, "idiopathic" or essential purpura, cases in which the cause is at present quite obscure.

Symptomatic Purpura.—Purpura may occur in a large number of the diseases of children. In one group it may be found towards the termination of a wasting illness such as chronic diarrhoea, tuberculosis, and inherited syphilis. Here the appearance of a purpuric rash, usually over the abdomen, as a rule indicates approaching death.

In a second group may be placed the purpura associated with various infections, such as scarlatina, diphtheria, varicella, variola, epidemic meningococcal meningitis, and erythema nodosum. Acute septicaemia may produce an extensive purpuric rash, and possibly also haemorrhage into the suprarenal glands. A common variety in this group is the term of slight purpura, usually confined to the limbs, in which there is a clear history of a recent sore throat. A child has a sore throat and develops purpura, and possibly some joint pains, probably due to some infection through the tonsils. This condition is commonly called peliosis rheumatica (Schödlers's disease), but is preferably termed "simple infective purpura" since it is very doubtful if it is truly rheumatic; probably the purpura is to be regarded as due to a separate infection occurring through the tonsils which are damaged by rheumatic tonsillitis. It is likely that the occasional appearance of this type of purpura in rheumatic children is to be accounted for in this way. The occurrence of purpura, except in connection with rheumatic nodules (p. 144) is extremely rare in cases of unmistakable rheumatism. Malignant endocarditis is a rare cause of purpura.

In a third group may be placed cases of purpura due to various blood diseases, such as acute lymphatic leukaemia, lymphadenoma, leucophila, and scurvy.

In diseases of the kidneys purpura occurs more readily in children than in adults. Jaundice, or rapidly growing sarcomata may be associated with purpura. In whooping-cough and epilepsy haemorrhages may arise from result of severe cyanosis.

Essential Purpura.—Inasmuch as the cause of essential purpura is unknown, the groups into which the condition has been divided are quite artificial. Where the skin only is involved the disease is spoken of as purpura simplex; where in addition there are haemorrhages into mucous membranes and joints, it is called purpura haemorrhagica (morbus maculosus Werlhofii); where abdominal cramps are present (to be mentioned immediately), it is named fleisch's purpura. Very severe cases associated with vomiting and unconsciousness, and running a fatal course of a few days only, are sometimes called purpura fulminans; it is probable that such cases at least are the result of an infection. Lastly, gangrene may occur as the result of the haemorrhages, especially of those in the mucous membranes (gangrenous purpura). This type is very exceptional.

Symptomatology.—The hæmorrhages into the skin may vary in size from small petechiæ to extensive areas of bruising. The mucous membranes most commonly affected are those of the nose and mouth, and from the epistaxis which may occur vomiting of the swallowed blood or mæconium may arise. In idiopathic purpura, retinal hæmorrhages are extremely rare, and when they are found it is a point in favour of the case being one of the symptomatic group. Bleeding into or around joints may occur and give rise to considerable pain. Hematuria is not uncommon, but actual nephritis is much less frequently present. The blood shows no distinctive changes in essential purpura.

The abdominal symptoms are of great interest. They are sometimes spoken of as "abdominal purpura" and form the distinguishing feature of Henoch's purpura. They are due to hæmorrhage into the walls of the intestine, chiefly into the submucosa. They tend to recur, a point of diagnostic importance. In a mild case there is some abdominal colic, with perhaps a little vomiting or diarrhœa. In severe cases there are agonising intermittent abdominal pains, with vomiting and the passage of blood and mucus by the bowel. The pain causes the patient to roll about in agony, and is somewhat relieved by pressure on the abdomen. Points which differentiate the condition from such inflammatory diseases as appendicitis or peritonitis. The hæmorrhage into the intestinal wall may be so copious as to give rise to a palpable tumour, and thus it will be seen both the symptoms and the physical signs closely simulate those of intussusception. Further, in rare cases the lumen of the gut is practically occluded by the large hæmorrhage into the bowel-wall, so that obstruction may be present. This, fortunately, is rare, but makes the picture almost completely resemble that of intussusception. The diagnosis in such a case is only to be made by the presence of obvious purpura elsewhere, or by a history of past attacks of purpura. At the same time it must be remembered that a localized hæmorrhage into the intestinal submucosa may actually give origin to an intussusception. The diagnosis from some disease of Meckel's diverticulum (p. 301) is one of great difficulty, and is to be made mainly by evidences of present or past purpura of the skin. Henoch's purpura may be mistaken for abdominal tuberculosis.

It is interesting to note that purpura is sometimes accompanied by erythema or urticaria, and that urticarial effusions into the wall of the bowel have been described, which may account for the attacks of abdominal pain seen in such diseases as these. Where no superficial and obvious lesions can be detected, it is a matter for speculation as to how often attacks of "colic" are due to abdominal purpura.

Prognosis.—In essential purpura the prognosis is good except in the cases of purpura fulminans. Even the abdominal symptoms, although so alarming, are not often fatal. In symptomatic purpura,

the grave significance of the purpura of marasmic infants has already been mentioned. In the acute specific fevers only the severer forms are associated with hæmorrhage. In leukaemia and lymphadenoma the occurrence of purpura is of bad omen. Hæmorrhages seem to take place far more easily in renal disease in children than in adults, and their presence does not add very materially to the gravity of the prognosis.

Treatment.—The treatment of essential purpura is at the present time very disappointing. A large number of drugs has been recommended, but so far as can be seen none is of particular value. The milder cases get quite well if they are kept in bed, but even strict rest does not prevent relapses occurring. In the more severe cases we are only able to treat hæmorrhage as it arises from certain parts of the body. The most dangerous form is epistaxis; this should be treated by the application of adrenalin to the nasal mucosa, and if necessary the nose should be plugged vigorously. Calcium lactate has been recommended, but in many cases the coagulability of the blood is not increased; if however such is found to be the case, calcium should be given. Ergot, iron, turpentine, and other drugs have been administered, but without satisfactory results; their use does not appear to stop the possibility of fresh hæmorrhages arising. For the abdominal pain, opium or morphia is generally indicated. Should it be necessary saline may be injected into the subcutaneous tissues or into the veins.

During convalescence the treatment proper to anæmia should be followed.

III.—DISEASES OF GLANDS.

I.—DISEASES OF THE LYMPHATIC GLANDS.

ADENITIS.

Simple adenitis, acute or chronic, does not need description here. Tuberculosis of the lymphatic glands, in so far as it is of medical interest, is described under TUBERCULOSIS (p. 129). Syphilitic adenitis is very rarely seen in the isolated form of the disease (p. 133).

LYMPHADENOMA.

Hodgkin's disease is not very uncommonly found in children under the age of twelve years, but differs very little from that seen in adolescents. It usually occurs in older children, but I have once seen it starting at the twenty-fifth month of life (Fig. 17, and 38). A case of extreme enlargement of the glands in lymphadenoma is shown in Fig. 50.



Fig. 37.—LYMPHADENOMA, STARTING AT THE THROAT. ENLARGEMENT OF CEREBRAL GLANDS.



Fig. 38.—LYMPHADENOMA, FIFTH OF THE MONTH. ENLARGEMENT OF SPLEEN.

2.—DISEASES OF THE SUPRARENAL GLANDS.

HÆMORRHAGE INTO THE ADRENALS.

This condition has been described by Binell in three groups of cases:—(1) Occurring before or during labour, due to injury, and causing death; (2) Occurring within the first few days of life, and probably due to a septiciæmia; (3) Occurring during the first few months of life in apparently healthy infants.

It is with this last group that we are here concerned. It is probably the least common of the three classes named. The symptoms come



FIG. 50.—CUSHING'S DISEASE.—GREAT ENLARGEMENT OF GLANDS.

on with extreme rigidity. A healthy infant is suddenly seized with severe abdominal pain. The screaming is quickly followed by severe collapse, and death occurs in a few hours. Purpuric spots may appear on the skin, and occasionally a tumour may be felt within the abdomen, due to the swollen suprarenal body. The origin of these cases is very obscure. It has been suggested that they are due to a toxin, and that there is a fatty degeneration of the walls of the suprarenal vessels. Some of them are probably to be classed with cases of "purpura fulminans" (q. v.).

ADRENAL SARCOMA.

In children this is not a very uncommon condition, and the cases strongly resemble each other. They occur in children of two years old and upwards.

The metastases are found in the glands and in the osseous tissue. As a rule the child comes under observation on account of swellings arising in the cranial bones. Growth in the roots of the teeth may cause proptosis, optic neuritis, and hemorrhage to the cellular tissue round the eyes. The brain vessel does not become involved. The ribs, clavicles, and sternum are usually the seats of secondary deposits, but these are generally externally placed, and so are not recognizable during life. The glands



FIG. 76.—JOSEPHINE TARKENTON, THREE YEARS OLD, IN FURTHER AND EXTENSIVE TO RIGHT SIDE.

throughout the body may become involved. The primary growth in the suprarenal gland becomes palpable as a rule before the child dies. The left side is affected more commonly than the right. The blood shows no characteristic changes.



FIG. 77.—JOSEPHINE TARKENTON, THREE YEARS OLD, IN FURTHER AND EXTENSIVE TO LEFT SIDE. (HISTOLOGICAL PHOTO COPY.)



FIG. 78.—JOSEPHINE TARKENTON, FURTHER VIEW OF SARCOMA THREE YEARS OLD, SUPRARENAL GLAND, GROWTH, MASS OF TISSUE IN EAR.

The pathology of the condition is not fully determined as yet. These tumours are at present classed as adrenal sarcomata, but it is probable that they are carcinomata of the suprarenal medulla (Farr, *Quart. Journ. Med.*, Jan., 1911.)

In another type adrenal sarcoma may be found in children of only a few weeks old, associated with sarcoma of the liver.

Diagnosis.—By the time the child is brought to the doctor the diagnosis is usually clear. By means of the abdominal tumour, asymmetry of the skull, and absence of any characteristic changes in the blood these cases are



FIG. 15.—ADRENAL SARCOMA.
APPEARANCE ON HEAD AT DEATH.

very easily distinguished from chloroma, lymphadenoma, and acute lymphatic leukaemia. The age-incidence of the disease serves to distinguish it from scurvy. The second type mentioned clinically resembles primary sarcoma of the liver (p. 110).



FIG. 16.—ADRENAL SARCOMA, DISTENTED BY FLUID.
(PRELIMINARY)

ADRENAL HYPERSECRETION.

A peculiar condition of physical precocity has been found in many instances (Ballou and Sigesbee) to be associated with either a hyperplasia of the adrenal

cortex, or a malignant growth, usually carcinomatous, of that organ (hypersecretion). A similar condition has, however, been found to be due to tumour of the pituitary or nasal glands, or of the ovary or testis and where the growth has been removed a return to the normal physical condition has followed operation. It would seem, therefore, that these organs act together in some way upon the processes of development. An abnormal condition of the adrenal gland, supposed to result in hypersecretion, is the most common



Fig. 15.—Precocious Obesity.

cause of the types of precocity about to be described, but occasionally no macroscopic abnormality is recognizable in any of the organs mentioned.*

Two types of this precocious development are known: that of precocious obesity, and that of precocious muscular development.

1. **Precocious Obesity** is found in both male and female children developing usually about the second year. The child becomes

* Dr. Leonard Gutherie, *Brit. Med. Jour.*, 1907, vol. ii, p. 747.

extremely fat, but the obesity is not in type that of childhood, but rather that of late middle-age. The face is bloated, the cheeks augmented, the belly pendulous, the mammae enlarged, and linear striae present on the abdomen and thighs. The trunk is as a rule more affected than the limbs. In addition, hair may be present upon the face and pubes and occasionally upon the rest of the body. Apart from the development of pubic hair there is as a rule no true sexual precocity, but occasionally it is present. These children are usually above the average in intelligence, but a few are feeble-minded or imbecile.



Fig. 76.—Precocious Obesity.

I am indebted to Dr. Gathre for two photographs (Figs. 75 and 76), which show well the bloated appearance of these children.

2. **Precocious Muscular Development**, the "infant Hercules" type, occurs in male children only. In this class a remarkable growth of stature and muscular strength develop without obesity. As in the previous group hirsuties may be present, but in addition there is precocious development of the sexual organs, together with that of sexual maturity.

Prognosis.—The outlook is bad in these cases. Death usually occurs at about the fifth year, and life is rarely prolonged to the normal age of puberty.

Treatment.—As has been mentioned where the origin of the abnormal development can be removed, as in the case of a tumour of the testis, improvement in the child's condition may occur. The adrenal growth is rarely palpable through the abdominal wall, but by means of exploratory laparotomy such an abnormality might be detected. Removal of the growth where present is as yet the only means by which a recovery is rendered possible.

ADRENAL HYPOSECRETION

Atrophy or destruction of the function of the adrenal body, or of the pituitary or pineal glands, testis, or ovary, may be associated with infantilism, and with non-development or loss of sexual attributes.

3.—ENLARGEMENT OF THE THYMUS.

Enlargement of the thymus is associated with two conditions of clinical interest. In one, thymic asthma, the enlarged gland compresses the trachea and gives rise to obstructive dyspnoea and possibly fatal asphyxia. In the second, that to which the term status lymphaticus is here limited, there are no such local symptoms and the condition is one in which sudden death arises in an apparently healthy subject from some trivial or even undetectable cause.

Thymic asthma is often included under the term status lymphaticus, an arrangement which, although theoretically reasonable, is not followed here owing to its practical inconvenience. The difficulties of the question are only farther increased by grouping under one title two classes which bear little clinical resemblance to one another.

Clinical Diagnosis.—The recognition of an enlarged thymus during life is difficult. A triangular area of dullness may be present under the manubrium. Its apex points downwards and reaches the level of the third rib, and its lateral margins extend beyond the sides of the sternum. The normal thymus during infancy does not give rise to this dullness, so that if present it may be due to an enlarged thymus or to tuberculous or other enlargement of the bronchial glands. Pallor in the interclavicular notch has been noted in some cases.

The most satisfactory method of detecting an enlarged thymus during life is by means of a skiagram.

THYMIC ASTHMA.

In this condition an enlarged thymus compresses the trachea. In very young infants the space between the manubrium of the sternum

and the vertebral column measures only 2 to 3 cm. and when the head is thrown back this is rendered still smaller.

Thymic asthma is described as arising in nurslings and as causing sudden attacks of dyspnoea, cyanosis, and stridor, which may be rapidly fatal. Cases have been recorded in which removal of part of the thymus has effected a cure, and in which the trachea at the operation has been seen to be compressed.

With this evidence the existence of thymic asthma cannot be doubted, but the condition is one of such extreme rarity that such a diagnosis could only be accepted if supported by very good evidence. In this connection two points must be mentioned. Firstly, post-mortem evidence is of little value, for the trachea unless it has been subjected to chronic compression, regains its normal shape as soon as the stenosis is removed. Secondly, a successful issue to an operation for removal of part of the thymus is of no positive value unless actual compression of the trachea can be made out at the time, since the administration of chloroform and the letting of a little blood will, as is well recognized, alleviate the symptoms in many cases of dyspnoea in infants. I have found the trachea perfectly intact in an autopsy upon a case of laryngeal diphtheria in which great relief had been given for twenty-four hours by what was thought to have been a tracheotomy.

STATUS LYMPHATICUS.

[*Lymphatism*]

It is only of late years that status lymphaticus has been recognized, and many more data are required before our knowledge of the subject can be considered satisfactory. Not only is the diagnosis of the condition during life practically impossible, but its recognition after death is a matter of great difficulty in children.

Symptomatology.—The subjects of status lymphaticus are usually fat, and may be anaemic, but do not of necessity present any symptoms of ill-health: indeed in most instances they are in apparently good health. The peculiarity of the condition is that in such subjects sudden death may arise from trivial or undiscoverable cause. The largest number of deaths attributed to lymphatism have arisen under anaesthesia, but in many instances death has resulted from the prick of a hypodermic needle, a plunge into cold or tepid water, or some other quite trivial cause.

Death is usually quite sudden, almost instantaneous, and appears to be due to syncope. In exceptional instances life has been prolonged for some hours or even days.

Lymphatism is usually regarded as a condition which persists from the earliest days of life, and while this is probably true of most cases there are apparently exceptions to the rule. Cases have been reported, for instance, in which death, attributed to lymphatism, has occurred

under anesthesia where anesthetics had been given on several previous occasions. Evidently some other view is required to explain such cases. It may be that lymphatism although persistent from birth is only at times dangerous and that death only occurs during a "critical period" (Humphry). On the other hand, such cases may be explained by assuming that lymphatism may sometimes be an acquired condition. Dr. Spillbury has recorded an instance in which the thymus showed microscopically both evidence of atrophy and signs of renewed activity.

Cases have been reported at all ages, from one year to fifty years, but are probably most common between the ages of twelve and twenty-two years.

Morbid Anatomy.—The post-mortem evidence of lymphatism consists in an overgrowth of most of the lymphoid structures of the body. The thymus is enlarged in nearly all cases but exceptionally may be atrophied. The glands at the back of the tongue and the tonsils are enlarged: in the spleen and intestine the lymphoid tissue is swollen. Adenoid vegetations are commonly present.

The myocardium is rarely normal as has been pointed out by Dr. Spillbury. The changes consist of fatty degeneration and brown atrophy, but are not commonly recognizable without microscopical examination. Their importance in connection with this disease is obvious.

The organs must be examined histologically in all cases. Particularly is this necessary in the case of the thymus, for what appears macroscopically to be an enlarged thymus may show under the microscope no real hyperplasia of the thymic tissue, the bulk of the organ being increased merely by deposited fat.

The lymphoid tissue shows histologically an increased activity as normal lym.

Diagnosis.—The diagnosis of status lymphaticus as the cause of death can only be reached by considering all the pathological changes together and excluding other possibilities. There is no doubt at all that in many instances death has been wrongly ascribed to this condition.

During life, as has been previously stated, an accurate diagnosis can hardly be made. Certainly the presence of enlarged tonsils and adenoids, and a moderate excess of lymphoid tissue at the base of the tongue cannot be said to be sufficient to warrant a positive diagnosis of lymphatism.

The enlargement of the thymus which is present in nearly every case requires special mention. The question naturally arises: What are the normal size and weight of the gland at various ages? It is because there is as yet no satisfactory answer to this question (owing to the variations in the size of the gland, apart from all considerations of lymphatism) that the recognition of the condition during childhood is so extremely difficult.

The natural variations in the weight and size of the thymus depend upon two chief factors: firstly, the age of the patient, and secondly, his state of general nutrition.

It is generally considered that the thymus under normal circumstances reaches a maximal size at the age of two years, remains practically stationary in size until the age of puberty, and then rapidly degenerates (Dodgson).

The other factor in producing variations in the size of the thymus is one which seldom receives the consideration it deserves, for it is one of extreme importance. The condition of the thymus depends to a large extent upon the general nutrition of the body. Where the child is much wasted the thymus is usually found much atrophied even in infancy; whereas in a fat child the thymus is fleshy and well-formed. From this follow two important considerations. Firstly, average figures for the weight of the thymus taken from a series of consecutive autopsies upon children, are of no value in determining the normal weight of the gland, since the great majority of such post-mortem examinations are conducted upon wasted subjects. Thus in the examination of 112 cases, Dr. Dodgson found that in ninety-five the thymus weighed less than 7 grams ($\frac{1}{2}$ oz.), while in the remaining sixteen cases, all of which died suddenly, the average weight was 25 grams (nearly 1 oz.). Secondly, since atrophy of the thymus and general malnutrition go together in children it follows that at an autopsy a thymus may be found to be "large" because the child has died suddenly (*i.e.*, has not had time to become wasted). In other words, the association between a large thymus and sudden death may be explained in many cases by holding that the thymus is large because death is sudden, instead of by stating that death is sudden because the thymus is large.

Probably the weight of the thymus in a normal well-nourished child is nearer 1 oz. than the more usually accepted figure of $\frac{1}{2}$ oz. More accurate figures are needed on this point before the normal weight can be dogmatically stated.

During the first few years of life the lymphoid structures throughout the body are in a much more active state than in an adult. The high proportion of lymphocytes in the blood and post-mortem evidence alike demonstrate this, and it is a point which adds considerably to the difficulty of recognizing lymphatism during the first years of life.

The possibility of regarding a fatty thymus as enlarged without microscopical examination has already been mentioned.

In conclusion, therefore, while we may readily accept the evidence for status lymphaticus as it occurs in adults, we must own that its recognition in childhood as an abnormal state is a matter of extreme difficulty and even of doubt, and that the differentiation of the abnormal from the normal is almost as difficult after death as during life, especially in children under four or five years of age.

SECTION X.

DISEASES OF THE NERVOUS SYSTEM.

I.—MENTAL DEFICIENCY.

General Etiology.—To some of the types of mental deficiency definite causes can be assigned, and to these allusion will be made later. We have here to consider what general factors may be responsible for the production of idiocy.

There are many popular ideas associated with the causation of idiocy, but it must be admitted that when approached scientifically, the subject is still one of great obscurity, and that we are not able to go further than to say that any circumstances which tend to weaken the reproductive power of the parents are likely to increase the chance of idiocy in the offspring. Neuropathic manifestations, alcoholism, and syphilis in the parents seem without doubt to predispose towards mental deficiency in the children. Consanguinity in the parents has been said by some authorities to be of no moment in the production of idiocy, but it is difficult to see how such a condition can fail to emphasise in the child any abnormalities, such as a neuropathic taint, present in the preceding generations of the family. Dr. Stoll has found that in 166 idiots of hospital practice, 24.2 per cent. were the children of first cousins; while Dr. Karl Pearson has estimated that the proportion of first-cousin marriages amongst the poorer classes is only 0.36 per cent. The place in the family is a matter of interest. Mangelson states, as is well recognised, tend to appear late in a family, and are frequently the last children born. On the other hand idiocy is very common in first-born children, and this is the absence of difficult labour or asphyxia. The birth of one idiot child does not predispose towards mental deficiency in later-born children, except in the case of anamnetic family idiocy, occasionally in macrocephalic idiocy, and in certain familial forms of cretinism not seen in England. That severe asphyxia at birth may cause irreparable damage to the nerve-cells of the brain seems likely, but cannot be proved. Instrumental labour is more frequent in the births of idiots than of normal children, according to Dr. Stoll's figures; but, as he points out, this is not necessarily a proof that injury by means of obstetric instruments causes the mental defect, it may be only that the difficult labour is likely to be accompanied by asphyxia. On the other hand, both

difficult labour and asphyxia may be not the causes of idiocy, but the signs of inefficient reproductive power on the part of the mother, whose uterine contractions are feebler than normal, and thus are likely to be present with greater frequency in the births of idiots than in those of normal infants.

The causes of acquired mental deficiency will be alluded to later.

Symptomatology.—The symptoms suggestive of mental deficiency may be described as positive and negative, of which the latter are the more important, but not as a rule the more obvious. In the earliest years of life, at all events, mental deficiency shows itself by acts of omission rather than by those of commission.

As has been already detailed (p. 21) the child, if physically and mentally healthy, in developing acquires certain powers at more or less definite ages. Failure to develop these functions at the right times may suggest, in the absence of physical illness, mental deficiency. Thus a baby should be able to hold its head up at the fourth month, to sit up at the ninth, and to walk by the eighteenth. If within three months after these dates, a child in good physical condition shows no signs of acquiring these powers, mental deficiency is probably present. Speech is a development subject to considerable variation in its time of appearance within the limits of mental health (see p. 467), but if by the eighteenth month no effort is made to say any word, mental deficiency is to be suspected. In idiots, habits of cleanliness are acquired very late, if indeed at all. Very obstinate constipation may in a few cases be the symptom which should give rise to the suspicion of mental deficiency.

Sudden, persistent, and purposeless screaming is often characteristic of idiocy. Apart from screaming, the noises idiots make are also purposeless, and do not correspond to the expressions of pain or joy of normal infants. But it must be mentioned that in some cases idiocy is accompanied by an absence of the usual crying and other noises of infancy. In later years idiocy is commonly associated with purposeless movements. These may be of an irregular nature, movements of the head, face, eyes, trunk, and limbs being perpetually present, which may be so marked as to cause an almost incredible degree of restlessness. Less often they are of a rhythmic type, and are seen in slow rocking movements of the body, or nodding or turning movements of the head, which persist for hours without abatement.

The tics is characteristic in some types of idiocy (described later), but often does not in the least suggest of itself any mental deficiency. Many idiot children, including some of the best hospital examples, are very pretty.

General Prognosis.—The prognosis in certain special forms of idiocy is discussed later. Here we need to consider the general lines of prognosis in cases of mental deficiency.

It not seldom happens that the physician is called upon to be the first to put before the parents the fact that their child is mentally deficient. Such a condition is often quite unexpected by parents, or is at the most a distant and dreaded thought. In such circumstances the truth must be broken to them as gently as possible. It is best to say that the child is "backward" or will never be quite so "intelligent as most children." "Mentally defective" is the strongest term that should be used, and the words "idiot" and "imbecile" should not be mentioned if avoidable.

Then come the questions, "Will the child live?" and "What will be his mental condition should he grow up?"

In reply to the first question we can only say that, excluding those types of idiosyncrasy (mentioned later) which are closely associated with an early death, the mentally-deficient child has a better hold upon life than has a normally developed one. This is particularly the case in the Mongolian type of imbecility, in which there are a large number of deaths from respiratory disease during the years of infancy and early childhood. Thus we cannot say more than that the chances of the child's reaching adult life are rather less than the average.

As regards the child's later mental condition, we must be prepared to answer such questions as, "Will he be able to walk, talk, look after himself in the world, and earn his living?"

The reply to these questions, if the child is seen in early infancy, cannot be made with any hope of its being absolutely accurate. We can only put before the parents the probabilities of the case.

The power to walk can be promised with fair certainty. It may not be acquired until very late, even until the age of five or six years is reached, but it is very exceptional to find that it is not acquired at all.*

The power to talk cannot be promised with such certainty. It may, however, be said that there is a great probability that the child will be able to talk. Speech will, without doubt, be acquired late, articulation is likely to be indistinct, and the vocabulary small, but in the majority of cases the child will be able to make himself understood.

Many other questions will be put to the physician, and where the patient is an infant we cannot give accurate answers to them all; in the absence of certainty it is our duty to take as hopeful a view as we can. To paint the future in the darkest colours is to be cruel to the parents, and perhaps to rob the child of its best chance of being rationally trained. We may say, therefore, that with education it is almost certain that the child will become clean in his habits, that he will be happy, able possibly to do little things from which he will gain pleasure; that he will be prone to be rather passionate and uncontrolled when

* Dr. Still, quoting Calkins, gives the prognosis of idiots who are unable to walk without assistance, as 10 to 20 per cent will walk, and 80 per cent will not.

amused, but will probably be affectionate, although jealous and suspicious. To suggest, however, that there is any chance of the child being able, when grown up, to look after himself in the world, take his proper social position, or earn his living, is unfortunately quite impossible; and if questioned closely on these points the doctor cannot do otherwise than put the matter frankly before the parents.

Nearly all mentally-deficient children make some improvement during the first few years of life, and if they are seen later a more accurate prognosis can be made. If there is then extreme fatuity, the child toying nothing and being quite heedless of his toys, the prognosis given can only be one to the effect that it is unlikely that much improvement will occur. The amount of improvement which has occurred is the guide to the ultimate condition of the patient.

General Treatment.—In the treatment of mental deficiency the question of education is all-important. The nurse must be quick to translate the slight signs by which the child expresses his wishes, so that in this he may be further encouraged, and she must be unwearying in her patient endeavour to force into the child's notice the common things of his life, and, by repeatedly naming them to him, teach him gradually to recognise and use the corresponding words. Again, the child must be taught to acquire control over his limbs, so that some day he may be able to put them to use. This may be done at first by means of toys, and later by exercises and drilling, often best accompanied by music. It must always be borne in mind that if a defective child can be taught to perform any little action expected of him, it is a source of no little gratification and happiness to him.

As a general rule the training is best carried out in some institution for the mentally deficient. There they are happier than at home. Mixing with children is a condition similar to their own and receiving the best training that can be had, they derive a good deal more pleasure from their existence than they do at home. Further, it not seldom happens that an idiot child is a source of harm to the other children of the family, especially if, as is often the case, they are of the neurotic type. Children of the hospital class are received into various institutions after the age of five, on their parent's application to the Guardians. There are now a few vacancies for children of the Metropolitan area at the earlier age of two and a half years.

Drugs, except in the case of cretinism (*p.* 441), play but a small part in the treatment of defective children. Sedative drugs, such as the bromides, chloral hydrate, and antipyrin, are of value in controlling fits, restlessness, and screaming.

Classification.—Imbecility is a term used indicative of a slighter grade of mental defect than idiocy. By a mentally backward child is meant, as Dr. C. West has put it, one who would be normal at an

early age, while for a mentally-deficient child it meant one who would be abnormal at any age.

Mental deficiency is usually considered under the two headings of Congenital and Acquired.

Congenital Idiocy may be classified into various groups: Mongolian, Cranium, Microcephalic, Hydrocephalic, Spastic, and Geriatric. These will now be considered.

1.—CONGENITAL IDIOCY.

MONGOLIAN IMBECILITY.

Dr. Langdon Down has given this name to a class of imbeciles which is easily recognizable on account of certain peculiar physical characteristics.



FIG. 77. (Mongolian Imbecility).—Eugen.

Etiology.—This group shows more clearly than any other the influence of the exhaustion of the maternal reproductive power. Mongols are very commonly found as the last born in a large family, and as the children of elderly women. The sexes are about equally affected. Mongols form, according to Dr. Still's figures, 22 per cent of the cases of mental deficiency in hospital classes, but at institutions for imbeciles only, those over 5 years of age being admitted, the proportion of

Mongols is at the most 1 per cent (Shuttleworth), a fact which suggests that the death-rate amongst these children is exceedingly high during the first few years of life. It is interesting to notice, as Dr. A. E. Garrod has pointed out, that Mongols are particularly prone to show congenital heart malformations, while complete transposition of the viscera has also been recorded in them. In a series of 52 Mongolian imbeciles Dr. Leonard Parsons found a congenital cardiac lesion in 36.5 per cent.

Physical Characteristics.—

These are recognizable at birth. The palpebral fissures are obliquely set, running downwards towards the nose (Figs. 77 and 78). It is because of this that the term "Mongolian" is applied to this class of imbecile, although it is well that these children do not in the least resemble a true Mongolian infant. In addition to the slanting eyes, the epicanthic folds are usually very well marked, while, as in other mentally-deficient children, squints are not uncommon.



FIG. 79.—MONGOLIAN IMBECILITY. HAND WITH SHORT, THICKENED LITTLE FINGER.



FIG. 78.—MONGOLIAN IMBECILITY. FAULTY MOUTH CHARACTERISTIC MOUTH EXPRESSION.

The cheeks are high-coloured, the bridge of the nose is depressed and broad. The skull is small, brachycephalic, and markedly flattened posteriorly. The skin of the face is often harsh and rough, due to salivation and the discharges from the nose and eyes, so that these children are prone, but elsewhere it is normal. The tongue is frequently protruded, and after infancy usually becomes much fissured. The palate may be high and narrow, but is often normal. The hair is normal. The mouth is small. Except during infancy the hands

are short and squat, but the tips of the fingers are tapering. The little fingers are often short and incurved towards the ring fingers (Fig. 79), but this peculiarity is seen sometimes in children of normal intelligence. As in other imbeciles, the circulation is often poor and the extremities cyanosed.

The stature is below normal, dentition is delayed, and the power of walking is acquired late.

Mongolian imbeciles are particularly prone to blepharitis, adenoid vegetations, and attacks of nasal catarrh, bronchitis, and bronchopneumonia. A large number die of pulmonary tuberculosis. The association of congenital heart disease and other malformations with this form of imbecility has already been mentioned.

Mentally, these children show some variation into grades of imbecility. As a rule they are happy-looking children, affectionate, often pained, easily irritated, mischievous, fond of noise, and showing a defective power of attention. They are restless, passionate and destructive. With careful education they can usually be trained to cleanliness in their habits. They learn to talk late, and in some cases can be taught to read a little, and to do simple work under supervision.

Morbid Anatomy.—No characteristic change is found in the nervous system of these children. The basal structures of the brain have been found unusually small in some cases, a change which may simply be due to the posterior flattening of the skull.

Diagnosis.—When the physical characteristics of Mongolian imbecility are familiar, the observer has little difficulty in recognizing the condition. The faces of inherited syphilitic show some superficial resemblance to that of a Mongol, but more often mistakes arise in regarding a Mongolian imbecile as a cretin. The chief points of differentiation are given in the following table:

	Mongol	Cretin
Age	Recognisable at birth	Not recognizable before 5th month.
Face	(Eyes oblique. Epicanthic folds marked. Cheeks high coloured.)	(Eyelids puffy. Nosebridge flattened out. Complexion sallow.)
Mouth	Small	Large, with thick lips.
Skull	(Small, brachycephalic. flattened posteriorly.)	(Oftent large and dolichocephalic.)
Skin	Normal.	(Hard and dry. Pads of feet.)
Hair	Normal.	Scanty.
Organs	(Eggs of teeth. Little finger short and starved.)	Separate teeth.
Stature	Below normal.	Much below normal.
Treatment by Thyroid Extract	No improvement.	(Much mental and physical improvement.)

Prognosis.—It is uncommon for a Mongolian imbecile to reach adult years. As has been mentioned, a large number die during infancy, and the majority succumb before adolescence. The diseases in which they are prone have already been enumerated, and are chiefly those affecting the respiratory system.

Mentally the condition is incurable, and, should the patient be seen during infancy, it may be stated that while the child will always be backward, he will learn ultimately to walk and talk, and may by training be made clean in his habits. That he could be taught to read or do any mental work cannot be promised, as such accomplishments as these are very exceptional amongst Mongols. In no circumstances, should the child grow up, would he be capable of earning his living.

Treatment.—In the poorer classes institutional treatment gives the child the best chance of happiness. There is, however, a slightly added risk of tuberculous infection where there are many such children together. Children in better circumstances may be trained at home. No drug treatment is of any special avail. The tendency which these children show to respiratory diseases necessitates the avoidance of the risks of colds or chills as much as possible. The nasopharynx may require attention.

CRETINISM.

Cretinism as it occurs in England is of the sporadic type, and differs from the endemic type of Switzerland and other places in Europe, in that it does not tend to run in families and is not associated with a goitrous swelling in the neck. Although probably an inborn condition, the symptoms are not congenital. It is considered here under congenital idocy, partly in obedience to custom and partly in order to place it next to Mongolism with which it may be confused.

Etiology.—The symptoms of the disease are due to the absence of the function of secretion on the part of the thyroid gland. As a rule the gland is absent, but it may be present, undeveloped or degenerated.



Fig. 86.—Cretin. (Age 4 years). (From . . .)

Physical Characteristics.—These are very clearly defined, becoming so, as a rule, during the second half of the first year of life. Before the sixth month cretinism is very rarely recognizable. The face is very characteristic (Figs 86-88). The palpebral fissures

are narrow, the eyelids puffy, the nose depressed, and the nostrils wide and splayed out; the lips are thick, the mouth large, and the tongue large, rounded, fissured and often protruding. The head often appears large for the body, = *dochecephalic*, and shows a few growths of coarse hair, usually light in colour. The complexion is *sallow*. The well-known pads of fat are a characteristic feature of the disease, except in infancy. They are most commonly situated above the clavicles and behind the sternomastoid muscles, but may be present at the axillæ or in other parts of the body. The skin is harsh, scaly and dry, and the extremities are often cyanosed. The temperature is considerably below normal, and is but little raised in the pyrexial attacks. The umbilicus is nearly always prominent, and is often the site of a hernial protrusion. Constipation is as a rule very obstinately present. The hands are short and stumpy, and the fingers are square at their extremities. All processes of physical development are much retarded; dentition and the closure of the anterior fontanelle are greatly delayed, and the lack of growth in the long bones is particularly noticeable, causing as it does great dwarfing of the child's stature.



FIG. 75.—CRETIN.—(Figs. 6 and 75.)

Under treatment these abnormalities, both physical and mental, are to a large extent improved.

Diagnosis.—A marked difference between a cretin and a Mongolian imbecile is given on page 438. Cretinism may also be mistaken for achondroplasia and some forms of staturalism, but as a rule with care there is no difficulty in arriving at a correct diagnosis.

Prognosis.—Without treatment there is no tendency towards improvement. With treatment the outlook depends to a large extent upon the age at which the disease is recognized, for it is obvious that the child coming late under treatment is handicapped by the loss of previous years. In addition it is probable that there are various grades of

continuum, so that all cases do not improve to an equal extent. The best results that can be hoped for are that the child on growing to adult years should be able to earn his living at some simple work, and that mentally he should be only very slightly below the normal. Possibly if the treatment were started in infancy, and faithfully continued, a total recovery might ensue.

Treatment.—The administration of thyroid to an infant is not devoid of danger. Over-dosage may result in irregularities of temperature, perspiration, a rapid and irregular pulse, or even fatal syncope. It is necessary, therefore, that the drug should be given at first with considerable caution. For an infant of six months an initial dose of one-sixth to one-quarter of a grain of thyroid extract (Burroughs, Wellcome & Co.) may be given once, and later, twice a day. Thereafter the doses may be rapidly increased, and so far as possible sufficient thyroid should be given to keep the temperature between 98° and normal. The treatment must continue throughout life. During the years of childhood, from one to two grains of the thyroid extract, given twice daily, constitutes the usual dose necessary for the maintenance of the treatment.

MICROCEPHALIC IDIOCY.

In many cases of idiocy the circumference of the head is below the normal, but this is not alone sufficient to warrant the inclusion of a mentally-deficient child in the clinical group of microcephalic idiocy. The characteristic of this group is not so much an absolute diminution of the circumference of the head, as a relative smallness of the cranium in proportion to the size of the face. By this disproportion in the growth of the cranium and face the forehead is caused to slope backwards in a characteristic way.

Embryologically this group shows no distinguishing features. Occasionally the condition is seen in several members of a family. Premature closure of the cranial sutures cannot be regarded as the cause of the maldevelopment of the brain, but is more properly looked upon as a result of the small size of the brain.

The physical characteristics are then those which have already been



FIG. 1. Microcephalic idiocy.

mentioned—namely, the sloping forehead due to the cranium being small out of all proportion to the face, and the petriovine closure of the anterior fontanelle. Occasionally spasticity is found in this class of idiocy.

Mentally every grade of backwardness, imbecility, and idiocy may be found.

The brain may show general maldevelopment, the whole structure being much diminished in size; or some parts of it may appear normal, while others are very rudimentary. Cortical sclerosis may also be found.

Treatment.—None is at any avail. Operations for removal of portions of the cranial vault formerly advocated are no longer practised.

HYDROCEPHALIC IDIOCY.

Little need be said of this form. It may be congenital or acquired (see p. 438), in which case it is usually the result of posterior bone meningitis. Care must be taken to make an examination of the mental condition of the patient, as very severe hydrocephaly may exist with very little impairment of the mental function. As regards treatment, none of the surgical operations which have been undertaken in these cases appears as yet to have met with any uniform measure of success.

SPASTIC IDIOCY.

This, although occasionally post-natal in origin, is usually associated with a congenital condition of cerebral diplegia (p. 474).

The mental deficiency is of very varying degree in this class. The patient is often subject to fits, but this tendency as a rule disappears as the child grows up and the mental condition shows some improvement. The power of speech is often very imperfect, but frequently the patient is able to perform simple work. A number of "village idiots" who hang about market-places are of this type. The mental deficiency is as a rule less marked in hemiplegic (usually post-natal in origin) than in diplegic cases. As already mentioned, some spasticity is occasionally present in microcephalic idiocy.

GENETOUS IDIOCY.

By this term is meant a congenital condition of idiocy which cannot be placed in any of the classes already described. These incommunicable cases form about 50 per cent of the congenital cases of idiocy. They show no distinguishing features, either mental or physical. Many show stigmata of degeneration, such as a high narrow palate, malformed and misplaced eyes, an abnormally shaped head, a face of low-grade intelligence, and many other abnormalities; but it is to be remembered on the one hand that such stigmata may be seen in children of normal

intelligent; and on the other that many grotesque idiots do not at first sight appear to be mentally deficient, but are bright-looking and pretty children.

2.—ACQUIRED IDIOCY.

In the great majority of cases there is considerable difficulty in obtaining proof that mental deficiency is an acquired condition, for certain evidence of a previously normal mental condition is seldom forthcoming.

ECLAMPTIC AND EPILEPTIC IDIOCY.

According to Ireland's distinction, eclamptic idiocy is the condition due to a severe bout of convulsions, and epileptic idiocy that resulting from the repeated attacks of chronic epilepsy. It is, however, very difficult to make certain that in any particular case the mental deficiency is a result of the convulsions, and thus to be able to disregard the possibility that the convulsions were merely associated symptoms of some congenital maldevelopment of the brain or of idiocy. Mentally-deficient children are very prone to epilepsy, and it is but seldom that one can trace with any certainty that the fits are directly responsible for the mental condition.

INFLAMMATORY IDIOCY.

This is the term given to mental deterioration acquired from some inflammatory conditions. Of this type, posthæmorrhagic (page 284) is one of the chief causes, and this disease may apparently pick out the frontal areas of the brain, and so give rise to mental change with no other permanent signs of cerebral mischief. On the other hand, certain cases of spastic idiocy, usually hemiplegic in type, should be placed in this group, as might also be some of the cases of so-called hydrocephalic idiocy.

Syphilitic Mental Degeneration occurs only in a few subjects of inherited syphilis. It usually conforms to the type known as juvenile general paralysis (p. 386).

IDIOCY BY DEPRIVATION.

Under this name are classified those cases in which, as the result of an illness, a child loses its sense of hearing, and as a consequence its ability to talk, and becomes mentally deficient. Such may occur in children up to the age of four or five years, and occasionally of an even later age.

TRAUMATIC IDIOCY.

This is a rare condition. Cases are frequently met with, it is true, in which mental deficiency is ascribed to a fall or blow on the head during infancy, but it is very seldom that there is any definite proof of the causal relationship of trauma to idiocy.

HYPERTROPHIC IDIOCY.

This is also a rare condition, in which the child, normal until one or two years of age, has begun to become dull, to suffer from headaches, and to show an abnormal growth of the size of the head. Such cases may also be congenital in origin. Death usually occurs before the age of puberty is reached. Pathologically the brain is increased in size, due to an excess of astrogia, without any evidence of hydrocephalus. This form of idocy can hardly be distinguished during life from the hydrocephalic type.

ANAUROTIC FAMILY IDIOCY.

Although a disease of very great rarity, this is nevertheless one of considerable interest, and one which constitutes on clinical grounds a well-defined entity. It is usually classed amongst the cases of acquired idocy, but while the symptoms do not appear at birth it cannot be denied that they develop as the result of some internal tendency.

The condition is sometimes spoken of as *Sach's disease*, or, more accurately, as *Wern's disease*.

Etiology.—Clinically, three points stand out clearly in this condition. First, it is practically confined to the Jewish race; second, it affects usually but not invariably all the children born to the same parents; third, it appears in one generation only. What the exact significance of these facts may be, or what the actual cause of the disease is, we do not know.

Symptomatology.—The first symptoms of the disease appear about the third to sixth months of life. It is noticed that the muscles of the neck are weakening, and as the child grows older this loss of power becomes more and more apparent. The limbs also begin to get weak, and as the disease progresses spasticity of the limbs develops. Then it is found that the child is gradually going blind, and as a rule starts at noises or on being touched, owing to this loss of sight. On examination of the eye-grounds double optic atrophy is found, but in addition a sign pathognomonic of this disease is usually present, consisting of a rounded, cherry-red spot in the position of the macula lutea, the retina surrounding this coloured spot being white and hazy in appearance. As the disease progresses the child wastes and as a rule death occurs during the second year after birth.

Morbid Anatomy.—There are usually no microscopic changes in the central nervous system, although in some cases the brain has been found to be unduly hard, due to secondary proliferation of the astrogia. Microscopically, however, evidences of universal cell degeneration, apparently primary in origin, are well marked. There is no sign of an inflammatory condition either acute or chronic, and no evidence that

syphilis plays any part in the causation of the disease. It appears to be a primary cell disease with secondary changes in the fibres. The most recently offered explanation of the changes found is that they are due "to some inherent biochemical property of the protoplasm of the cells"; they do not correspond to those of pure atrophy. The ganglion cells of the retina share in the changes, and it is from the extreme thinning of the retina (possibly its actual perforation) at the macula that the vascular thread is seen in this area as the typical cherry-red spot.

Diagnosis.—The diagnosis of the condition is easy if the possibility of the disease be remembered. Confusion may arise from ticks, cerebral diplopia, and posterior basic meningitis. The macular changes are distinctive of amaurotic family idiocy.

Prognosis.—This is uniformly bad, death being inevitable, and usually occurring about the second year of life. The outlook for any children born later in the family is also extremely bad. It is exceptional for any to escape.

Treatment.—None has hitherto been of any avail in preventing the fatal issue of this disease.

II.—FUNCTIONAL NERVOUS DISORDERS.

THE NEUROTIC CHILD.

Before proceeding to the study of the various functional nervous disorders found in children, it is necessary to consider the type of child who is subject to such conditions.

Dr. Leonard Goffine* describes two main types of neurotic children: (1) *The unrestrained emotional type*. (2) *The restrained emotional type*.

1. **In the Unrestrained Emotional Type** the emotions are strong but ill-controlled; the energies are excessive but ill-restrained. The child is as a rule thin, pale, and ringed darkly round the eyes. The appetite is capricious, the action of the bowels irregular. The attitude tends, if the child feels well, to be one of constant restlessness, the fingers moving, the head quickly turning from side to side, the eyes glancing from object to object. If feeling out of sorts the child becomes pale and limp. The mother will usually describe her child as having "highly-strung nerves" but as being easily exhausted. So too with the mental state. The mental balance is long-armed, but so delicately poised that the most trivial factor produces an excessive response. Gaiety, affection, attention to work, these tend to be excessive, and to

* *Functional Nervous Disorders in Childhood*.

change, without instantial cause, to misery, unbridled indignation, and cloth, in their turn excessive and unreasoning. In these children the imagination is superabundant, and their care for facts is a minimum. There seems neither sad distress there, and later the mechanical nocturns of Latin verse wrangle them. Such things "won't come right" with them.

2. **The Restrained Emotional Type** shows superficial differences. Here again the emotions are strongly felt, but the power of control is also strong. Such children are serious and reticent, have no great liking for rosy games, and show but little affection, and as a consequence are often thought sullen, cold-blooded and wanting in affection, when such in truth is by no means the case. These children are so reticent and reserved that they are not able to break through their shyness and claim that affection for which in reality they are longing.

In the first type the nervous energies of the child become exhausted as the result of the physical and mental restlessness and instability, and there is a tendency for various functional disorders to be developed. Night-terrors, somnambulism, headaches, habit-spasms, hysterical palsies, hysteric diarrhoea, enuresis, asthma, are commonly noted in these children, while should they become infected with rheumatism, they inevitably show signs of chorea. Born with this temperament the character they acquire shows the traces of lack of self-discipline and control. In the second type, that of restrained emotionalism, the nervous energies also become exhausted, but here on account of the unending and severe restraint that is shown. Such children may develop the disorders already mentioned, but are prone to various secret fears (phobias), attracted to things horrible, yet morbidly afraid of them.

Etiological Factors.—For the proper treatment of neurotic symptoms in children, nothing is of greater importance than to try to ascertain their cause and to do what can be done to remove it.

An inherited neuropathic tendency is probably the most important factor underlying the production of functional nervous disorders. Without this the child's nervous system is far more stable than where it is present. But acting upon this inherited condition are many and various factors which predispose to the development of nervous disorders, and these are of great importance, as they may be capable of alteration or removal. The circumstances of the child's home may have a bad effect on him; with neuropathic parents he is likely to have not only a bad inheritance, but a bad upbringing. In neurotic children various sources of peripheral irritation may determine the onset of some disorders, as may be well seen in some cases of habit-spasms and hysterical pandemonium. Certain mental factors may often be recognizable. Of these great fright, over-work (or more often over-worry about work) are the most frequent. Illnesses of all sorts will lessen the stability of the nervous system, but there are in children two

dances which stand in particularly close relationship to various functional nervous disorders:—

The first is rickets. In infancy such conditions as laryngospasm, strabismus, tetany, and facial irritability are seen practically only in combination with rickets, while the same disease is certainly a predisposing factor in the production of convulsions and the condition known as head-nodding and myasthenia.

The second is rheumatism, and the association here is so intimate as to require some consideration. Rheumatism will produce a disease in which definite or severe mental and motor phenomena are present, which condition we call chorea. Although it is no longer possible to regard chorea as a "functional" disease, yet it is certain that, given a rheumatic infection, chorea is more easily developed and less readily cured in a neurotic than in a non-neurotic child. This is only what is to be expected, and this consideration explains at once the facts that chorea is nearly three times as common in girls as in boys, that in adults it is practically confined to the female sex, and that past evidence of a neuropathic tendency is very frequently found in choreic children or their antecedents.

When we were considering chorea (*op. cit.*), we saw that it was not a disease of disordered movements alone, but one showing both mental and motor symptoms; and here we must endeavour to trace these symptoms back to the earliest point at which they become recognizable. On the motor side these earliest signs will be seen as generalized fidgetiness, together with some clumsiness in the finer voluntary movements (such as sewing). In addition there will be fleeting facial movements over-expressing the emotion of the moment (the "waxy smile" of these children is very characteristic), and possibly irregular respiratory movements, as shown in the frequently repeated long-drawn sigh, so often heard in neurotic women. Mentally, the earliest changes are easily recognizable, and consist for the most part in loss of control. Thus excitability, inattention, outbursts of passion, easily excited attacks of crying, and morbid fears are usually found. Frequently the child's mother has noticed the change in disposition, and complains that she (for it occurs most often in girls) is now too frightened to sleep in the dark or to cross a crowded street.

We see therefore that the earliest signs of chorea are not more than would give rise to a diagnosis of general nervous instability, and this condition of rheumatic origin we perhaps may conveniently term "latent chorea," to distinguish it from the fully-developed disease. It is seen in three sets of cases: (1) In association with obvious rheumatic manifestations, as synovitis, nodules, and heart disease; (2) In children convalescent from such rheumatism or from chorea; (3) In children who show only slight symptoms of the rheumatic infection. The first two classes are described on p. 446.

It is with the last group that we are here particularly concerned. It is of great importance that it should be recognized that a rheumatic

infection may produce very definite nervous instability without actually proceeding as far as the condition we call chorea, that it does so more easily where by inheritance the arthritic temperament is present; and that the symptoms proceeding from this nervous instability may and frequently do cause the child's visit to the doctor. In all cases, therefore, of children of this type, it must be made a rule to examine for the slightest manifestations which are recognizable as definitely rheumatic, such as tonsillitis, growing-pains, stiff-neck, pain in the side or epigastrium, headaches, or heart disease; for should we miss the essentially rheumatic nature of the case, we may allow the heart to become severely and permanently affected.

There is one further slip. As rheumatism is a cause of general nervous instability, so it may be the basis of nervous disorders far removed from chorea. Of these night-terrors, semicomatose, habit-spasm, henteric diarrhoea, and nocturnal enuresis are the most common, and in such cases as these every precaution must be taken to avoid missing the rheumatism which is responsible for their appearance.

It is not for a moment suggested that all nervous children who suffer from these disorders, and are pale and show slight dilatation of the heart, are in reality rheumatic; but it is most necessary to emphasize that rheumatism may be, and in London very frequently is, the cause underlying such conditions.

NERVOUS DISORDERS ASSOCIATED WITH RICKETS.

Three such disorders are found almost invariably associated with rickets: these are tetany, laryngismus stridulus, and facial irritability. Crepitation, and the condition known as head-nodding and myasthenia, are more commonly found in rickety children than in the non-rickety. Thus these conditions are all closely associated clinically.

TETANY.

(*Carpopedal Spasm*.)

Etiology.—Tetany or carpopedal spasm is most frequently found in infants, and is in them almost invariably associated with rickets, and often with the nervous disorders just enumerated. In addition, in the great majority of cases some gastro-intestinal disorder, usually diarrhoea, is present; and dilatation of the stomach or some other part of the alimentary tract can often be demonstrated.

Tetany in older children has been described by Dr. Langmead (*Clinical Soc. Trans.*, 1897) in association with dilatation of the large intestine (p. 283). In this group there is no evidence of active rickets. The tetany is very prone to recur, and is sometimes associated with facial irritability and laryngismus stridulus.

Transient tetany is seen on very rare occasions in older children with acute gastro-intestinal indigestion.

The exact origin of the symptoms of tetany is not yet fully understood, but some toxin absorbed from a disordered or distal alimentary tract appears to be the cause of the condition. Whether the parathyroid bodies have any action in producing the tetany of infants is not known.

Symptomatology.—The symptoms are characteristic. There is a tonic spasm of the muscles of the limbs affecting chiefly the hands and feet (Fig. 81). The hands are held in a characteristic position (accoucheur's hands) in which the fingers are extended while the metacarpophalangeal joints are semiflexed, as are, in bad cases, the wrists and elbows. The thumb is drawn into the palm and frequently protrudes between the second and ring fingers. The feet are plantiflexed, the toes rigidly flexed. The spasm remains for several days. At its first appearance there is a good deal of pain, but this may pass off before the spasm is completely relaxed. In this disorder the dorsal surfaces of the hands and feet are often swollen, redematous, and may be pruritic. This swelling is usually accounted for by saying that the spasm interferes mechanically with the venous return from the parts, but inasmuch as these swellings are absent in some cases of tetany and exactly similar swellings are found in cases of diarrhea without tetany, it seems likely that in many cases, if not in all, the edema is toxic in origin. The febrile symptoms found in tetany as it occurs in adults, do not appear to be present in infants; indeed, they can be held to account for some of the cases of sudden death among infants with tetany. In rare instances the spasm may affect the muscles of the trunk.



Fig. 81. Tetany (1). Contracted Spasm in a Case of Unusual Abundance of Lactal Secretion.

Trousseau's sign requires mention. It is brought out by encircling and compressing the arms near the shoulder, so as to put pressure upon the brachial nerves and vessels. Within about half a minute the hand assumes the position of tetany if the infant has recently had an attack. The affected muscles show increased electrical excitability, and, as life desecrated, the muscle shows contraction becomes greater than the cathodic.

Prognosis.—This in infantile cases depends upon the general state of the patient, the gastro-intestinal condition being of special importance, but sudden death in cases of tetany is not very rare. The spasm itself can usually be relieved in a few days.

In the tetany of older children with a dilated large intestine, although the attack may be relieved there is a great tendency to recurrence, and a fatal issue is to be expected within a variable period of months.

Treatment.—We have to relieve the spasm, to treat the gastro-intestinal tract, and to deal with the rickets underlying the condition. For the spasm opium is the best drug, and serves in addition to alleviate the pain which may be present, and to control diarrhoea. Chloral hydrate and the bromides are also of value. In the treatment of the alimentary tract lavage is of the greatest service, and should be employed if there is any gastric dilatation or persistent diarrhoea. The good effect of lavage is well seen in the cases with dilatation of the large intestine, where this treatment will cure the tetany for the time being. When the acute symptoms have subsided, and the digestive tract has been put in order, the rickets should be treated by cod-liver oil and dietetic measures.

LARYNGISMUS STRIDULUS.

This is practically confined to rickety infants, and may be associated with tetany and facial irritability. It consists in a spasmodic closure of the glottis without any underlying catarrhal condition. The infant, apparently at good health, suddenly "holds its breath," and becomes livid. After a few seconds the spasm relaxes, and air is drawn into the lungs with a loud crowing inspiration. The attack is then over, but is liable to be repeated later. Between the attacks the child is well and suffers from neither hoarseness nor cough. With the attack there is no stridor, no dyspnoea, but rather a temporary apnoea, respiration being for the time stopped. The spasm may be brought on by excitement, and it may be thought that the child is merely in a passion. Exposure to cold may bring on the spasm, or it may occur as the child awakes from sleep. In slight cases little is noticed apart from the "crowing."

Diagnosis.—We have to differentiate the condition from one in which the spasm is superadded to laryngitis, either catarrhal or membranous (p. 335). The matter is usually settled easily, for in laryngismus there is no cough or hoarseness, no stridor, no inspiratory recession of the chest, and as a rule no fever, while between the attacks the breathing is easy and quiet.

Prognosis.—The outlook is good, but it must be remembered that very rarely death does occur from laryngismus, so that it is not a condition in which there is absolutely no danger, as is often assumed.

Treatment.—During the spasm this may consist of applying a hot sponge to the throat, or, should the attack be severe, the finger should be introduced into the mouth, and the fauces stimulated as though to produce vomiting. Immersion in hot water or the administration of chloroform by inhalation will allay the spasm, but in the first attack these measures are seldom to hand; they may be held in readiness for future occasions. If the spasm be severe and the child become weak and cease to struggle, artificial respiration should be undertaken at once, and those in charge of the child should be instructed how to carry this out.

The intestinal condition should be attended to, castor oil or calomel administered, and the action of the bowels regulated by glyster powder or rhubarb. The bromides, chloral or belladonna, may be given for a period of a few days, until the nervous system is quieted. A diet rich in fat should be ordered as soon as the state of the digestion permits, so that the underlying richest may be benefited.

FACIAL IRRITABILITY. (Chvostek's sign.)

This consists of a slight contraction of the muscles at the corners of the eyes and mouth, produced by tapping the facial nerve in front of the ear over the par. Auricular. It is merely an indication of the irritability of the nervous system found in rickety infants, and it may be associated with tetany, laryngismus, or convulsions. Except as a guide to the condition of the nervous system, the so-called Chvostek's sign is of no moment. The method of eliciting the sign is shown in Fig. 24.



FIG. 24.—FACIAL IRRITABILITY AND TETANY.
Showing the method of eliciting Chvostek's sign.

CONVULSIONS.

Etiology.—Convulsions, as they occur in infancy and childhood,

are to be regarded as due to a large extent to the instability of the nervous system, and are therefore particularly prone to occur in the subjects of ticks. Although the unstable condition of the nervous element is probably the chief factor underlying the convulsive tendency, yet many reflex causes may be at work.

It is an important point to remember that in an infant a convulsion associated with reflex irritation may not be generalized, but remain unilateral—that is to say, that a unilateral convulsion in an infant may not indicate any local disease or injury of the brain, and must not be regarded as of any localizing value.

(1). Of the reflex causes which may set up convulsions, the most common are those acting in the alimentary tract, particularly constipation. Improper feeding and indigestion of all sorts may occasion convulsions in children whose nervous systems are unstable. Morbid conditions of the respiratory system are liable to produce similar results, as may pain from such conditions as pinworms, or painful dentition.

The association between dentition and convulsions is one of such great importance to many minds that a parent's attention must be given to it. Teething is popularly supposed to account for so many and such alarming states that if only an infant is "about his teeth," attacks of fever, convulsions and other conditions are cheerfully allotted to him as his right, and teething is still regarded as the cause of as many maladies as formerly were worms.

When in later life the eruption of the tooth causes pain, it is due to an over-crowded state of the jaw, and under normal conditions the physiological action of cutting a tooth is unaccompanied by pain. In an infant, therefore, the same thing should hold good, and the gum should be absorbed over the erupting tooth without discomfort. How is it then that teething has come to be looked upon as the source of so much illness? Most are agreed that where dentition is painful the gums are in an unhealthy condition, and it is to be remembered that during infancy the mouth participates to a peculiar extent in the morbid changes associated with disease of both the alimentary and respiratory systems. We see then that the mistake so often made, in the case of rickety infants especially, that the child runs his teeth with diarrhoea or bronchitis, is looking at the matter in the wrong light. It is because the child has some natural condition of the intestine or bronchi associated with swelling and tenderness of the gums, that the dentition becomes painful and so attracts attention. While we know that diarrhoea alone will produce convulsions, yet where painful dentition is also present the tendency to convulsions is increased.

The term "several convulsions" is used for the condition, often the result of colic, in which the child shows some rolling of the eyes, stiffness and crying, without any actual twitching of the muscles or loss of consciousness.

(1) Apart from the reflex convulsions which we have been considering, fits during childhood may be due to epilepsy, to gross meningeal or cerebral disease, or to the onset of some acute infection.

The diagnosis of epilepsy without organic disease of the brain can hardly be made during infancy, and, should the question arise, as it often does, as to whether the child will or will not become a confirmed epileptic, although no definite answer can immediately be given, a good prognosis is as a rule justified. Only about 10 per cent of infants who have convulsions become epileptics later, so that as a whole the outlook is distinctly good. A neuropathic family history, stigmata of degeneration, severe and long-lasting convulsions, and a total absence of any discoverable underlying or reflex causes, tend to make the development of epilepsy in after years more likely. At about the fourth year the diagnosis can usually be made. Mental deficiency is much more often a precursor than a result of convulsions.

Organic cerebral disease of various forms may be responsible for or associated with convulsions. Mention may be made of cerebral sclerosis (the cause of most cases of cerebral diplegia), of chronic meningitis, and the destructive changes of acute encephalitis.

The onset of bacterial infection is not uncommonly associated with a convulsion, which in the opinion of some corresponds to the rigor in an adult. In addition, convulsions frequently terminate cases of wasting in infants, and are often accompanied by hyperpyrexia. Tonic convulsions are rare in children, except in scurvy.

Prognosis.—The outlook in convulsions depends for the most part upon the type of the disease present. Where the convulsion is due to reflex causes acting upon an unstable nervous system the prognosis is quite good. With the presence of gross cerebral disease the condition is very much more serious. The outlook with regard to future epilepsy has already been dealt with. Where the onset of an acute encephalitis is associated with convulsions and unconsciousness lasting for many days, there is great likelihood of permanent damage to the brain being present to such an extent that mental deficiency, with or without epilepsy, may ensue.

Treatment.—The indications for treatment are to cause the cessation of the fits, to remove the source of any reflex irritation, and to endeavour to prevent recurrence of the symptoms.

For the fit itself immersion in a hot bath may be of temporary use. Chloral hydrate and bromide are the most useful drugs to employ for a sedative effect. Of the two, chloral is the more efficient. Both are well tolerated by children. They may be given per rectum if necessary. Inhalations of chloroform may be requisite in severe cases until the action of the chloral asserts itself.

Of the many reflex causes of convulsions in infancy, the commonest

a gastro-intestinal dyscrasia, and an emema and a full dose of castor oil or of calomel should be given; and, as measures are taken to restore a healthy condition to the alimentary tract, the tendency to convulsions will cease. Other causes should be carefully looked for, such as enlarged tonsils, adenoids, pleurisy, intestinal parasites, tender gums or abnormal conditions of the eyes or ears.

In order to prevent recurrence of the fits, the rickets, so often present, should be treated, and for a time the child may be given small doses (2 to 3 gr. for an infant) of bromide.

HEAD-NODDING WITH NYSTAGMUS.

(*Spasmus Nutans*).

This is a condition which is usually associated with rickets. It occurs as a rule between the ages of 6 and 12 months, and very regularly begins during the winter months of the year. The nodding or rolling movements of the head go on incessantly unless the child's attention is distracted. They are varying in rate, usually about once or twice a second. As a rule, they are accompanied by nystagmus, although often this does not develop until movements of the head have been present for a few days. The nystagmus may be vertical, horizontal, or rotatory; and the movements of the eyes are much quicker in rate than those of the head. There seems to be no truth in the view that the two sets of movements are in any way compensatory. Occasionally, the eyes are steady, unless the head is fixed; although, as a rule, the eye-movements are thereby increased. It is worth while noting that the nystagmus may be more marked in one eye than in the other; and, indeed, may be quite uniaxial.

Of the *etiological factors* at work is the production of spasmodic nutans, the presence of rickets is probably the most important; but, in addition, reflex irritation from the bowel or elsewhere may be present. The theory that the movements are caused by cystitis, due to the child living in a dark room, can hardly be regarded as proved.

Diagnosis.—The diagnosis of true spasmodic nutans rarely presents any difficulty; but somewhat similar movements of the head may be produced as a result of acute middle-ear inflammation. The movements of head-rolling and head-lounging are of a different type (p. 453).

Prognosis.—The outlook is quite good. Although the symptoms may not disappear for several months, the condition is entirely without harmful effect.

Treatment.—Any possible source of reflex irritation should be removed; as a rule the best results are obtained on the lines of general constitutional treatment. Sedatives, such as bromide, may be given, but their benefit is rather doubtful.

SCREAMING.

Screaming in infants is not to be regarded as invariably due to hunger; indeed it is more frequently the result of over-feeding. Should the screaming be wrongly attributed to hunger, the condition is likely to go from bad to worse, so that care should be taken not to allow the child to be fed at irregular intervals whenever it cries. Thirst is a frequent cause of screaming, and may easily be managed by the administration of a little water. Flatulence, colic, and cold feet are causes of screaming which should not be overlooked. Plumbism or hyperacidity of the urine are likely to cause screaming associated with the act of micturition. Very severe screaming on being handled, in an infant of six to twelve months of age, should suggest scurvy rather than meningitis. Where excessive screaming continues as the child grows older, mental deficiency may be suspected.

HABIT-SPASM.

(*The Tics*.)

The term habit-spasm is one which has for long been given to one of the commonest functional nervous disorders of childhood, in which there is a certain oft-repeated movement of a muscle, or of groups of muscles. The name is more convenient than accurate, and many now apply the term "tic" to this condition. By considering simple, co-ordinated, convulsive, and psychical tics, although no fine lines of distinction can be drawn between these groups, we can include all the forms of the disorder.

Etiology.—According to Dr. Still's figures, 75 per cent of the cases start between the fifth and tenth years of life, while the disorder is only slightly more common in girls than in boys. Apart from these considerations there are, however, a number of etiological factors of interest.

Of first importance is a neurotic temperament. The children who are the subject of habit-spasm are those of the "unrestrained emotional type" (p. 445), and need not here be described. Given the unstable nervous system, any cause rendering it still less stable will be predisposing towards this disorder. Of such, the most important are: fright, worry, excitement, fatigue, intestinal parasites, indigestion, and all diseases which exercise a temporary debilitating effect. The deleterious influence of rheumatism on the nervous system, whereby a condition of nervous instability or of latent thoria is set up, has already been discussed (p. 447). The importance of this infection in predisposing towards habit-spasm is well shown by Dr. Still's statistics. In one hundred consecutive cases of habit-spasm rheumatism was found in the patient, or his antecedents, no fewer than thirty-seven times. The

relationship of tics to habit-spasm is thus seen to be very close; that between habit-spasm and chorea is mentioned below, under diagnosis. Lastly, one habit-spasm predisposes towards others.

There are then many factors in the production of the irritable and unstable nervous system which is the basis upon which habit-spasm is grounded, but it is difficult to say what determines the onset, or the particular nature of the disorder. The presence of some local irritation can be proved in some cases, and in many more may be suspected; but how often this is actually the cause of the tic (in a neurotic child) cannot be stated. It is quite certain, however, that should the habit-spasm be started by local irritation, it may remain long after the irritation has passed away. Thus it is to be expected that in many cases due to this cause the actual source of the irritation cannot be traced. The most common of these are: conjunctivitis, errors of refraction, carious teeth, chronic nasopharyngeal or laryngeal catarrh, enlarged tonsils, and adened vegetation.

Symptomatology.—There are innumerable varieties of tic, but they show several points of similarity. In the first place, the movement is the same each time it is repeated; it may be repeated quickly, or at long intervals, or it may occur many times in rapid succession, and then cease for many minutes, but it is the same movement for days together. In this it differs markedly from the movements of chorea, which cannot be foretold. Secondly, when the patient is under direct observation, the tic is as a rule in abeyance; often it does not show itself unless the child is led to believe that he is not watched. Here, again, chorea differs entirely from habit-spasm. Thirdly, any one habit-spasm predisposes to any other. As a rule the first is discontinued when the second develops; but the two may occur together, the second being added to the first.

The commonest of all habit-spasms is the twitching or screwing-up of the eyelids. Twitching of the nose or of the corner of the mouth, frowning, sniffing, coughing, rolling-up of the eyes, are very common forms of tic. Such movements as these often replace each other.

More complicated movements may be present (*co-ordinata* tic). The most common, perhaps, is a turn of the head towards a shoulder, which is at the same moment elevated, or a sudden affirmative nod of the head. The limbs may also be affected, the arms more often than the legs. All sorts of various movements, simple and complicated, may be met with. In comparison to the movements are of the same kind, but more sudden and jerking in character. *Psychical* tic includes such disorders as the sudden utterance under impulse of obscene phrases (*coprolalia*), or of peculiar imitative notes,

Diagnosis.—As a rule there is no trouble in recognizing a habit-spasm, but occasionally a difficulty arises in connection with chorea; not is this to be wondered at when we consider the relationship of

rheumatism to habit-spasm. It has already been pointed out that an infection by rheumatism causes nervous instability, and so predisposes towards habit-spasm. Should the rheumatism produce a greater upset of the nervous system, chorea will result. It is often difficult to say whether a child is merely neurotic or actually choreic; and this is so whether a habit-spasm is present or not. Further, one may often see a child affected with a tic develop chorea, or, conversely, a habit-spasm may become grafted upon a residual chorea. By the consideration of a rheumatic nervous instability (latent chorea), the association between habit-spasm and chorea is best explained. A tic cannot be regarded as directly due to a rheumatic infection, as is chorea; but it may be the outcome of a rheumatic infection through the latent chorea which this induces. In such cases the rheumatic basis of the tic should be treated. The movements of a habit-spasm and of chorea differ in that in the former they are off-repeated, similar movements, which are lessened when the patient is watched; whereas in chorea the movements are unexpected, cannot be foretold, and are increased by inspection of the child. Exceptionally a tic is made worse by observation.

Prognosis.—As a rule this is good, but it must be remembered that a habit-spasm is a symptom of a profoundly neurotic temperament and not itself a disease, so that there is a great tendency for a tic to recur in one form or another. In this way some children are afflicted with various tics for several years.

Treatment.—We have to treat the habit-spasm itself, and the underlying nervous instability. Any source of local irritation, if such can be found, must be removed, but such a procedure as an operation for the removal of adenoid vegetations may upset the child so much that the tic is made worse by it. In some of the cases in which movements of the head occur, it seems that the long hair hanging round the neck and over the eyes may be a source of irritation. At all events some are benefited by having the hair tightly plaited in an unbecoming style. Scolding, punishments, and promises of reward are all of no avail, and may even be harmful; nevertheless the child should be encouraged as far as possible to control the impulsive movements.

The neurotic temperament of the child will require careful treatment. Removal from school and from other causes of mental excitement is a very important step, which is both necessary and beneficial in most cases. A few, however, do better at school than at home, owing to parental and home circumstances. In obstinate cases it is best to send the child into the country amongst strangers. Nothing is more beneficial than this total change of environment.

In very young children, the movements are more due to mimicry than to a true tic, and it is usually wisest to ignore them stolidly. If they fail to attract attention they generally cease spontaneously.

Drugs play only a minor part in the treatment of hicc. Where there is reason to suspect a condition of latent toxæmia underlying the habit—poor, salted and alkali should be given until pains, irritability, and headaches have been got rid of. Apart from this, bromide, phenazone, and arsenic are of use in reducing the mental excitement of the child; while other measures, such as cod-liver oil and iron, or those which will correct dyspepsia or constipation, may be of value.

HEAD-ROLLING.

This is most commonly found in rickety infants under the age of two years. The child rolls its head persistently from side to side as it lies on the pillow, until the hair on the back of the head is worn away. As a rule there is no painful condition to account for this, which is simply a habit, but care must be taken not to overlook some inflammation in the ears. This form of head-rolling is newly distinguished from spasmodic infans, in which the movement of the head lacks the voluntary appearance of that in head-rolling. It usually passes off by the end of the second year of life.

HEAD-BANGING.

This disorder occurs at a later age than the foregoing as neurotic or eccentric children. The child bangs the head against some object, or hits it with his fists. It is most commonly seen to occur in outbursts of temper. No harm is done by this rather alarming habit, as with head-rolling, this head-banging may be associated with pain in the head, from the ears or brain, but such is exceptional.

BODY-ROCKING.

This is another habit of neurotic or eccentric children. They will sit slowly swaying to and fro, often accompanying the movements with a creaking noise. Such movements in female children may originate or be a sign of masturbation. In the latter case the movements will be associated with excitement and followed by exhaustion. There is, however, no necessary association between the two habits.

ECLAMPSIA NUTANS.

This name is given to a condition of head-jerking occurring in children. The movements are sudden and jerking in form. They occur in a series of fifty or more, and are accompanied by a dreamy state of consciousness, or even temporary unconsciousness. Allied to this is the condition known as caliam fits, in which the body is suddenly bent forwards, the hands being extended before the face, with their palms downwards. Both these conditions—*eclampsia nutans* and *caliam fits*—are forms of epilepsy.

MASTURBATION.

This is not an uncommon habit in nervous children, and may be practised even in infancy. It is more common in little girls than in boys. It may be done by rubbing the sexual organs with the hands, but in small girls is more often the result of "thigh-rubbing." In this the thighs are tightly crossed and the child rocks itself to and fro or "wriggles." Sometimes such movements as these are done in a sitting posture. Occasionally, one leg is bent and the heel of the foot pressed against the vulva. Whatever method is adopted, there are the characteristic accompaniments. The child becomes excited and flushed, perspiration breaks out, and is followed by pallor and exhaustion. The habit may thus be mistaken by the child's parents for some form of epilepsy.

Masturbation is due, in part, to the neurotic temperament of the child, but in many cases some local irritation, balanitis or vaginitis, may set up the habit by the itching and discomfort caused. An attractively nurse, or bad companions at school, may likewise be responsible for teaching the child the habit.

The results of masturbation have been scandalously exaggerated, and more harm has been done by the false teaching about this condition than was ever done by the habit itself. There are no grounds for supposing that insanity is ever the outcome of masturbation practised by a previously healthy child. The number of cases in which mental disease is even indirectly attributable to self-pollution is extremely small (not more than 2 per cent). The condition is a symptom, rather than a cause, of nervous instability. The symptoms which have been said to be due to masturbation are simply those of neurasthenia, and the habit of self-pollution cannot be said to produce more physical harm than any other cause of exhaustion. Through the habit of coeunt which it tends to foster, it is productive of mood rather than of physical or mental deterioration.

Persistent masturbation practised openly is usually a sign of mental deficiency.

Treatment.—The treatment in infancy is, as a rule, easy. The child must be interrupted in the process, and though this needs a little firmness on the part of the parents, it is easily accomplished. The strictest attention to the cleanliness of the sexual organs is necessary. In older children the habit is not so easily broken, partly because it may have been practised secretly for a long time, particularly in the case of female children. Again the treatment of any local source of irritation is of great importance. In addition, the child must be under strict supervision. Punishments should not be ordered, as they are likely to give rise to deceit on the child's part, but rewards for abstention from the habit for some stated interval may have a good effect. If

necessary, force must be used to prevent the child from practising masturbation, and no chances for secret indulgence should be allowed. The child should be told that such a habit is naughty, and that it ruins him, and renders him unfit for games and the livelier forms of gaiety; but in no circumstances should he be made to feel himself an outcast as the result of his practices. Such a feeling is likely to make him depressed, morose, and introspective. It is preferable to explain to him that he must conquer this habit, which is wrong, and to show a desire to help him to do so.

Drugs may be of some avail; mechanical restraints is seldom of more than temporary use. The bromides and belladonna are of service. Tonic drugs may be ordered; but of more value are exercise in the open air, and a general active out-door life.

NIGHT TERRORS.

(*Pavor Nocturnus*).

Much has been written on the differences between nightmares and night terrors, but no definite line of distinction can be shown between the two. We cannot do more than say that what is a nightmare to a normal child is a night terror to a neurotic child. Nor can we divide night terrors into symptomatic and idiopathic. Probably all are symptomatic, though the cause cannot be traced in each case. The more neurotic the child the smaller and less easily discoverable will be the cause of the night terror.

The complaint is found in children between the ages of three and eight years. The symptoms usually arise within the first three hours of sleep. On a sudden, piercing screams are heard proceeding from the child, who is discovered sitting up in bed, pale, shaking, with staring eyes gazing intently at some part of the room, wherein evidently is the imaginary object of his fright. He may, however, have left the bed, and be crouching in a corner, shielding himself with his hands, or may have run shrieking out of his bedroom. He clings for protection to any one who goes to him, but recognizes neither the person nor his surroundings at first. After a few minutes (up to half an hour) the child finds himself again, and sinks back in bed pale and exhausted. He will ask not to be left alone, and after a little time goes off into a sleep, at first uneasy, later normal. The terror is not repeated on the same night. After the attack he may pass a large quantity of urine of low specific gravity; but often incontinence has occurred during the terror.

Hallucinations of sight are those usually present in a night terror, as may be recognized by the child's cries of fear; but those of hearing and common sensation may also occur. Not uncommonly the hallucinations are the same in each attack.

The next day, as a rule, the child knows quite well what has been

the subject of his fright; but occasionally there is either no recollection, or the child is too frightened to talk about the horror of his dream.

When we come to consider the etiology of night terrors, we cannot trace in all cases the reason of the particular form which the terror takes, any more than we can explain the basis of dreams in each instance. Much, however, is known. The most important factor is the neurotic temperament, and all the conditions, inherited and acquired, which lead to nervous instability are those associated with the production of night terrors. As much emphasis has been laid on the importance of latent chorea (p. 447), it is interesting to note that Goodhart found a rheumatic parentage in 17 out of 37 cases of night terrors, and has drawn attention to their frequent occurrence at the onset of chorea (regarding them, however, as a possible cause of chorea*). While agreeing as to their occurrence early in chorea, I would prefer to regard them rather as a symptom of nervous instability due to latent chorea.

The actual exciting causes of night terrors acting on a neurotic child cannot, as has already been said, be always traced. The most common are probably indigestion (especially constipation), enlarged tonsils and adenoid vegetations, producing partial asphyxia while the child is asleep. Similarly stuffy rooms, bedclothes too heavy or too tightly tucked in, excitement or fright during the day, hypersensitivity of the urine, and pain of any sort may induce night terrors.

The relationship of night terrors to epilepsy is important. As night terrors are symptomatic of nervous instability, it is only to be expected that some cases should show a family history of epilepsy, should have had convulsions in infancy, or should develop epilepsy later. Inasmuch as an epileptic fit may be brought on by severe fright, it is to be expected that a night terror will, in occasional instances, immediately precede a general convulsion. Although rarely a night terror may possibly be a form of epilepsy, especially when it is associated with day terrors, there is no proof that this is the usual relationship. So far as we know the great majority of night terrors have nothing to do with epilepsy.

Treatment.—This follows the usual lines of dealing with functional nervous disorders. The child must be placed in conditions of mental quietude and physical good health. Salicylate of soda should be given where there are definite evidences of recent rheumatism, combined with 4 doses of bicarbonate of soda or chloral hydrate at night. In other cases, the sedatives alone may be given throughout the day. The avoidance or treatment of the various causes enumerated above, is of great importance.

* Goodhart and Staff, *Disease of Children*.

DAY TERRORS.*(Panic Infinites.)*

Of less frequent occurrence than night terrors is this somewhat similar condition. The fear comes upon the child during the daytime. He is, however, conscious of his surroundings and calls loudly to the nearest person for protection. Much the same hallucinations are present here as in night terrors.

The relationship of day terrors to epilepsy seems nearer than that of night terrors, and in some cases they seem closely allied, if not to epilepsy, to hysterico-epilepsy and narcolepsy. It has also been suggested that they are due to vertigo or migrain. The influence of the various sources of peripheral irritation is not so clearly seen as in night terrors. The significance of day terrors is graver than that of night terrors, no doubt partly owing to the fact that a greater degree of nervous instability is necessary for their production.

Under treatment (as for night terrors) these attacks, as a rule, pass off rapidly.

SOMNAMBULISM.

Sleep-walking is not uncommonly found in neurotic children. Dr. Still has noted its occurrence in rheumatic families. It is easily amenable to treatment on the lines laid down for the treatment of night terrors.

PICA.

Pica, or dirt-eating, is usually found between the ages of one and two years, but may persist until long after the second year of life is completed. It is a habit confined to neurotic and eccentric children (excluding imbeciles). The patient will eat all forms of dirt, such as earth, cabbage-leaves, soap, pieces of brick, and for such things as these there seems to be a craving, ordinary food being refused. In addition to the neuropathic state of the child, there is usually indigestion and constipation, in part the result, but possibly in part also the cause, of the habit. The child wastes, becomes sallow and unhealthy in appearance, and suffers from some abdominal pain.

In a somewhat different category comes the swallowing of rather odder children. Such may lead to intestinal obstruction, and cases are on record where the stomach has been opened and a large hair-ball removed with success.

Pica is, as a rule, easily curable. The digestion must be set right by a course of aperients, and the appetite improved by tonic drugs and fresh air. The child must be watched, to prevent its getting any of its favourite materials for eating. In addition, it may be wise to send the child to the seaside for a time.

TEETH-GRINDING.

Teeth-grinding is a common habit in nervous children. It occurs nearly always during sleep. Its occurrence during the day is rare, apart from idiosyncrasy or gross brain disease.

Teeth-grinding may be due to meningitis, especially in the posterior base font, when it occurs in children past the age of infancy. Much more commonly it is due to some peripheral irritation acting upon an unstable nervous system. The commonest of these causes are dentition (secondly, carious teeth, ear-diseases, and constipation with or without the presence of worms).

The treatment of the habit is sufficiently indicated by the enumeration of the causes of its appearance.

HEADACHE.

In children headache is a common complaint, and may be due to a number of causes. Of general causes, rheumatism and arthritis (often rheumatic neuritis) are the most common. Goodhart found that in 33 cases of headache, no fewer than 25 were of rheumatic tinct. Headache as an early sign of chorea has already been emphasized in dealing with that disease. Local causes are most commonly found in the eyes (hypermetropic astigmatism), nasopharynx, carious teeth, or disease of the ears. Various forms of indigestion, especially constipation, are frequent sources of recurring or persistent headache. As in adults, gross intracranial disease is associated with headache; in children incipient tuberculous meningitis has especially to be remembered. Uremia is an uncommon cause in children. Typical migrainic may occur in children, and, as has been mentioned, day ferries are held by some to be due to a migrainous aura.

Treatment.—Little need be said here, for, as a rule a cause for the attacks of headache can be traced, and treatment should be directed towards that. The headache of rheumatism is very amenable to treatment by salicylate of soda.

EPILEPSY.

Etiology.—In children epilepsy, associated with gross intracranial disease, may be due to meningitis, acute and chronic, intracranial abscess or tumour, congenital malformations of the brain (e.g. sclerosis), acute polyencephalitis, and other conditions. It may also be due to trauma, uræmia, and general bacterial infections.

Epilepsy without organic nervous disease should be divided into two groups: (1) idiopathic, and (2) symptomatic, reflex or accidental. The distinction between these two classes is of great importance, as Dr. L. Guthrie has emphasised.

In true idiopathic epilepsy no cause can be traced for the fits, which are attributable to some inherent vice of the brain. The patient is usually dull, and below average intelligence; and the prognosis, both as regards the cessation of the fits and the ultimate mental condition, is poor; and in children between the ages of two and twelve years the outlook is very bad.

In symptomatic, reflex, or accidental epilepsy, the nervous system of the patient is also unstable, but not to a degree sufficient to give rise to epileptic discharges apart from some stimulus, physical or chemical, which produces for the time further instability. Thus, from physical ill-health, or mental worry, the irritability of the brain may become more marked, or the epileptic discharge may be set up by peripheral causes from the eyes, ears, teeth, alimentary tract, or genitourinary organs. The patient in this group of cases is highly neurotic, ill-controlled, and unstable, but is usually of a mental capacity of or above the average. Here, then, the prognosis will tend to be better than in the former class; but the smaller the stimulus causing the epilepsy, the less favourable is the outlook.

The importance of this subdivision is well seen when we examine the prognosis of all cases of epilepsy (excluding organic cases) in relation to the ages at which the disease first appears. In infancy the outlook is good, only about 10 per cent. of the cases becoming confirmed epileptics. Here, obviously, the proportion of reflex cases would be high. But in epilepsy starting between the ages of two and ten, the outlook is very bad (as it is later in life, from 25-35). This is a period of life when, to a normal brain, the stress of life should be slight; there is but small trouble, as it were, for epilepsy, and, should it arise, the danger of a confirmed habit resulting is great. During puberty and adolescence (the opposite holds good), the stress of mental and physical development is then great, and the prognosis is comparatively good.

It must, however, be remembered, that in a case from some reflex cause, the irritability of the brain may become so great by the repeated epileptic attacks, that the case may pass into what is practically an idiopathic epilepsy from having acquired the "habit" of epilepsy.

Symptomatology. *Grand mal* in children is similar to that in adults. Mention has already been made of the fact that a reflex convulsion in an infant, due usually to some intestinal derangement, may be entirely unilateral and not general.

Petit mal in children may be easily overlooked. It may, for instance, be regarded as a fainting attack. As a rule, a faint can be distinguished from *petit mal* by the fact that consciousness is lost gradually in the former and suddenly in the latter. In neurotic subjects, however, where the brain is very unstable, consciousness in a fainting attack may be lost extremely rapidly. Dr. A. L. Russell, on the other hand, has reported cases in which typical fainting attacks, with gradual onset, have become more and more sudden in type, and have ultimately

developed into true *petit mal* and *grand mal*. It is obvious, therefore, that it is far from easy in all cases to be sure of the character or significance of a "fainting attack." Where such occur, associated with nervousness, anemia, and slight dilation of the heart (often due to rheumatism), it may be fairly certain that the attack is not epileptic in the usual sense of that term. Again, where the child is repeatedly having slight attacks of loss of consciousness, the diagnosis of *petit mal* becomes clear. Between these two definite types are many cases which are difficult to interpret.

Again, *petit mal* may be mistaken for periods of inattention, the child apparently being in a "brown study," for attacks of violent passion, or quiet silliness, or extreme disobedience. The association between epilepsy and day terrors has already been mentioned (p. 462). Some cases of diurnal rectal incontinence are due to *petit mal*. There is a good rule to the effect that in cases of extraordinary functional nervous disorders in children, epilepsy should be suspected.

Repeated attacks of *petit mal* are likely to develop into *grand mal*, and to be associated with mental deterioration.

Diagnosis and Prognosis of epilepsy in children have been sufficiently discussed above. In true idiopathic cases a cure may be expected in probably less than 10 per cent of the cases.

Treatment.—It is of first importance to ascertain any possible reflex cause for the condition, and to treat that. While this will do but little good in idiopathic cases, unfortunately common in children, it may do much for symptomatic cases if they come under treatment early. In addition, we should endeavor to quiet the brain by the use of bromides and to restore its equilibrium by changes in the mode of life and mental activity, diet, and tonic drugs.

In accidental epilepsy bromides are needed, as a rule, only in small doses, the attention to the general health of the patient being of greater value in rendering the cerebrum less irritable.

In idiopathic epilepsy the bromides may easily be given in too large quantities, and means for improving the patient's general health neglected.

Children take bromide very well, and the drug may be used in exactly the same way as with adults. The diet should be full but plain and wholesome, while over-eating should be guarded against.

PSEUDO-PARALYSIS.

In children there are many conditions which cause apparent weakness or paralysis, apart from an organic nervous lesion or a hysterical condition. Most commonly pain is the cause of a pseudo-paralysis. Movements of a limb may cause great pain, and so the part is kept absolutely immobile.

Thus, in infancy, syphilitic epiphyseal scurvy, and acute osteomyelitis, will be the most common causes. In older children, rheumatism, acute osteomyelitis, or hemorrhage into joints (as in purpura or haemophilia) may give rise to a pseudo-paralysis. Tuberculous disease of joints, owing to its more gradual onset, less often gives rise to this symptom.

Perhaps the most common cause of a child "going off its legs" is rickets. This is a pseudo-paralysis, but in rather a different category from the group already mentioned. In this there is definite weakness, but at most only slight pain; the muscles are flabby and hypotonic. The child begins to get worse in its walking, until soon it refuses to put its legs to the ground at all, drawing them up and crying when held out to walk. When it is lying down, however, it kicks its legs about well. Evidently the legs have become too weak to support the child's weight, although they are not paralyzed. There is some pain, probably, when the child attempts to walk, and this is most likely to be due to straining of tendons and muscles. Any actual deep hyperesthesia of the legs is uncommon; if found, it is probably due to the presence of some scurvy element in the condition.

The differential diagnoses of these conditions and their treatment have been given in other parts of this book.

FUNCTIONAL PARALYSIS.

As it occurs in children, this is, as a rule, the result of some painful condition of the affected part. It is not very uncommon for a child to refuse to use a limb the joints of which have been painful from rheumatism; or, again, during a traumatic attack the muscles of the back may have been painful, so that when cured the child will make no effort to sit up. Indeed, any condition which has given rise to pain may, in a nervous child, cause disinclination to make those movements which so recently were a source of discomfort. In a functional paralysis of one leg the gait is often very characteristic, the foot being dragged along with the dorsal surfaces of the toes touching the ground. The gait may show some peculiar form of ataxia. Often the child will make no effort to support himself on his legs, and, when put to the ground will collapse with the legs under him.

In another group of cases there may be some weakness of a limb due to an organic condition; but to this may be added so much functional exaggeration that there is grave danger that the whole condition may be regarded as of functional origin. For instance, an early sign of an intracranial growth may be slight weakness of a limb, but as the result of functional exaggeration the limb may appear entirely paralyzed.

Another cause of functional paralysis, which is occasionally found, is neuritis. A child, usually of a very nervous type, who has

been associated with some one suffering from a paralytic deformity, they develop an exactly similar condition.

When the child is taken away from its usual surroundings, placed with healthy children, and treated firmly, the trouble as a rule, rapidly subsides if no actual organic basis for the symptoms is present. Occasionally, however, particularly in the last group of cases, the malady may be very stubborn and resist treatment for a long while.

DISORDERS OF SPEECH.

Before considering the various forms of faulty speech, we must discuss the common condition of delay in the acquisition of speech.

DELAYED DEVELOPMENT OF SPEECH.

The age at which the instinct of speech first appears is subject to considerable variation within physiological limits. As a rule, by the end of the first year a few words are spoken, such as "mum" and "dad-dad." The number of words becomes greater during the following months, until, at the eighteenth month, short sentences of a very elementary type may be heard. By the end of the second year the child should be able to name a large number of common objects, and to talk fairly well.

Delayed development of speech may be due to a variety of causes, and the cases may be divided into the following classes:—

1. **Simple Delayed Development of Speech.**—In this condition no mental or physical defects are to be found; the instinct to speak is simply late in appearing. The patient is quite intelligent, and feels that, by pointing at what he wants, he can obtain his desires without speaking. In such children speech may not make its appearance until after the second year, and instances have been reported where no articulate words were spoken until even the fifth or seventh year. Such children show no signs of mental deficiency: they learn to sit up and to walk at the proper ages; they understand all that is said to them, and express themselves by intelligible signs. Speech, when it does appear in them, usually develops rapidly.

2. **Delayed Development of Speech due to Mental Deficiency.**—This is, perhaps, the largest group of cases; and some mental defect should be suspected in all cases where no signs of speech being acquired have appeared by the age of eighteen months. The diagnosis is made by the absence of those signs of normal mental development mentioned in the preceding paragraph. The prognosis as regards speech being ultimately acquired is good, although the full power of speech is not likely to be entirely developed (see p. 424).

3. **Speech Defects due to Deafness.**—Deafness may give rise to various forms of speech defect. In the first place, the patient may not be entirely deaf; but, the hearing being indistinct, the development

of speech is delayed and when acquired, articulation is imperfect. Words are imperfectly heard and imperfectly reproduced. Secondly, absolute deafness may give rise to deaf-mutism. The deafness may be acquired or congenital, and rather over 50 per cent of cases of deaf-mutism are due to acquired deafness (Still). Loss of the power of hearing before or shortly after the development of speech causes non-development or loss of that faculty. In young children acquired deafness is usually due to double otitis media; occasionally to postencephalic meningitis. Congenital deafness (congenital deaf-mutism) is very prone to be found in several members of a family.

With careful educational methods, the child can be taught to lip-read and to speak; and these should be started at as early an age as possible.

1. **Congenital Word-deafness.**—Strictly speaking, there is no deafness here, but, as the child resorts to some extent to deaf-mutism, it is perhaps advisable to mention the condition in this place. To all spoken language the child appears quite deaf, taking no notice of what is said, and making no attempt to speak; but that he is not deaf is seen at once by his appreciation of musical or other sounds, apart from those of speech. Even if he can be got to repeat the sounds of words, they convey no meaning to him. It is evident, therefore, that the auditory centre receives correct impressions, but that there is a congenital absence of the means of connection between this centre and the parts of the brain concerned in the interpretation of the sounds heard. Such cases as these are apparently more common than was formerly imagined. The children are necessarily backward, but not mentally deficient. Careful training should be started as soon as the condition is recognized in order that the child may be taught to connect certain things which he sees with certain noises (words) that he hears.

LOSS OF SPEECH.

It is not common for a child who has acquired speech, to lose it again, except as a result of the loss of the power of hearing, as has been mentioned. In a neurotic child temporary loss of speech may arise from fright, and rarely may follow some severe illness. More commonly, loss of speech is a symptom of chorea, and is usually associated with severe paralytic symptoms. It may last for as long as nine months, but will always ultimately get well as the chorea passes off.

Loss of speech from the gross cerebral lesions, such as may be often found in adults (haemorrhage, embolism, etc.), rarely occurs in children.

"STACCATO SPEECH."

This disorder is occasionally found in children, and may be due to cerebral encephalitis (p. 274), or to Friedreich's ataxia (p. 477).

DEFECTIVE SPEECH.

We have here to consider:—

- (1). *Stammering and stuttering* (inco-ordinate speech, *dyslalia*);
- (2). *Talking and lisping* (defective articulation, *paralalia*);
- (3). *Idioglossia*.

1. **Stammering and Stuttering.**—Unfortunately these terms have been used in different senses by different authors. Popularly, stammering and stuttering are synonymous terms, and are applied irrespectively to all forms of inco-ordinate speech (*dyslalia*). Some authors have, however, distinguished between stammering and stuttering, using the former to denote a "spasmodic arrest of utterance," and the latter, "spasmodic repetition of initial syllables of words" (Guthrie). With these, therefore, stammering and stuttering are both forms of inco-ordinate speech (*dyslalia*). But the German authors confine the use of the term stuttering to all forms of inco-ordinate speech, and by stammering denote the forms of defective articulation (*paralalia*).

In the short description that follows, the terms stammering and stuttering are used as synonyms, to denote inco-ordinate speech.

Etiology.—Slight stammering is normal at moments of excitement in children who have only lately acquired the power of talking. Morbid stammering occurs after fluent speech has been acquired, and most commonly arises at, or soon after, the sixth year. Boys are more commonly affected than girls. Stuttering is a functional disorder, and dependent upon a nervous temperament, and thus may be associated with other functional conditions—as night-terrors, enuresis, halit-spirans, and such disorders. Anything which temporarily increases the instability of the nervous system (e.g. fright, exhaustion, worry, illness) will predispose towards stuttering, and may originate or increase the severity of the disorder.

The actual defect itself arises from two factors, both affecting the muscles of articulation, phonation, and respiration—namely, inco-ordination and spasm. The fact that quon is necessary explains why stuttering is not seen properly in choirs, where inco-ordination is so marked.

Symptomatology.—There are various types of stuttering, the difficulty being usually most marked in the articulation of consonants, and only occasionally concerned with the vowel-sound production. The faults of speech occur usually at the beginning, and rarely at the termination, of words or sentences.

The first letter of a word may cause the difficulty, the child being unable to sound it, or he may repeat it, or the whole of the first syllable, several times before being able to finish the word. Less commonly an initial vowel-sound forms the obstacle. Spasm of the respiratory-muscles is sometimes seen in the sudden inspiration which is taken just before speaking, and which causes a delay.

The mechanism of speech is briefly as follows. Firstly, the movements of the respiratory muscles, about which the most important fact is that expiration should be fully controlled by means chiefly of the diaphragm and abdominal muscles. Thus, as large or as small a volume of breath as is required is expired. Secondly, the movements of the vocal cords, by which vowel-sounds are produced, to be varied by the modifications of the cerebral chamber through which they pass. Thirdly, the movements of the lips, tongue, and soft palate, by which the various consonants are produced by interruption of the expiratory current.

Sometimes, the action of these parts is perfectly co-ordinated; in stuttering it is spasmodic and unco-ordinate.

The sounds of speech, then, are produced by obstruction to the expiratory current of air, and this obstruction occurs at various points:

- (1.) At the lips, as in producing *b, p, m*.
- (2.) At the tip of the tongue, as in producing *t, n*.
- (3.) At the back of the tongue, as in producing *k, g* (hard), *y*.
- (4.) At the larynx, as in producing phonation for vowel-sounds.

These positions are called the first, second, third, and fourth "stop" positions respectively.

From these considerations it can easily be seen where the chief difficulty arises in any particular case of stuttering.

Treatment.—The following general principles may be laid down. The child's health must be brought up to as good a level as is possible. He should nearly always be removed from school; the timid child is made worse by the unsympathetic treatment he receives at school, and the stolid and indifferent boy will not endeavour to make any improvement while at school. Any abnormalities of the nasopharynx or mouth should receive attention.

The particular fault in each case must be ascertained. By reading-exercises it can be found out at what "stop" positions the difficulties arise.

In most cases the initial fault lies with the management of the breathing. This is shown by the facts that stuttering does not usually occur in singing or whispering, and less often in reading verse than in ordinary speech.

The child should be encouraged to speak both slowly and quietly.

The various exercises for the cure of stuttering are seldom carried out under the immediate guidance of the doctor. As a rule the routine to be employed is something as follows:—The patient is first taught to breathe regularly and deeply, and particularly to gain control over the exit of his breath by means of the diaphragm and abdominal muscles, rather than by closure of the glottis. Having acquired this, he then learns to introduce vowel-sounds into his expirations. Lastly, he learns to introduce the various consonants into the vowel-sounds which he intones, learning particularly to introduce the

consonants without interrupting his pronunciation. The object of treatment is to make the mechanism of speech a co-ordinated habit, and until it has become a habit there is danger of relapse.

2. **Defective Articulation (Paralalia).**—In this condition the child substitutes an easy sound for one he finds difficult to utter. In young children this is normal; in older children, abnormal. One form of paralalia consists in substituting "th" for "s," "u" for "i," or "j" for "r"; but in other cases quite different substitutions may be made for various sounds, while many sounds may be habitually omitted. Such a condition is very common in tubercle children, but is by no means confined to them.

3. **Idioglossia.**—This term has been given to a group of cases in which paralalia is so extreme as to render the child's language quite incomprehensible. The child talks fluently, makes use of the same sounds to represent the same words, and, consequently, appears to be talking a language of his own. Although the condition is found chiefly in children of neurotic substance, they are not mentally defective. They are, of course, likely to be backward, owing to the difficulty experienced in their education. Idioglossia does not mean an invented language, nor an atavistic tongue, but is simply an extreme example of paralalia, all difficult sounds being altered or omitted. Possibly there is, in some cases, an imperfect perception of sound on the part of the child. Dr. Gelline has noted total absence of nasal ear in such a patient. In a case under the author's care, in which articulation had been improving, a temporary attack of deafness caused a very noticeable but transient deterioration.

Treatment.—Of first importance is the prevention of defective articulation. Children learning to speak should be taught to articulate words clearly, and not be encouraged to pronounce words incorrectly. "Baby-words" must, of course, be learnt; but even these should be pronounced properly. When the habit has become fixed, much may be done by careful training in reading and singing. In the extreme cases (idioglossia) cultivation of an ear for musical tones is much to be recommended. It is not always wise to put the child with other children in order that he may be cured, for not seldom the others acquire the defective articulation, and the patient remains unimproved. Idioglossia rarely persists after the age of puberty.

III. ORGANIC NERVOUS DISEASES.

Organic nervous diseases will be considered in three main groups:—(1) *Cerebral*; (2) *Spinal*; (3) *Peripheral*.

1.—CONGENITAL NERVOUS DISEASES.

OBSTETRICAL PALSIES.

Facial Paralysis is the commonest birth-palsy. It is in most cases due to the pressure of the forceps upon the peripheral part of the facial nerve. It may also be due to the difficult labour which has caused the use of the forceps, and occasionally a slight facial paralysis occurs where delivery has not been instrumental, the damage being effected, probably, by the pressure of the external promontory on the nerve. The lesion is nearly always unilateral, but complete on the affected side. The face on the paralyzed side has the appearance of being swollen, and when the child cries, the characteristic appearance is seen. The tongue is unaffected, and this swelling is not much interfered with. The prognosis is good. The paralysis, as a rule, clears up in about a fortnight. It is rare for it to last for a long period, and still rarer for it to be permanent. The amount of damage done to the soft parts of the face is only a rough guide to the severity of the nervous lesion. In treatment, nothing beyond keeping the eye on the affected side particularly clear need be done for the first fortnight. If the paralysis has not improved by that time, gentle rubbing of the face, or even electrical massage, may be begun.

Erb's Paralysis.—This is a much more serious birth-palsy than is facial paralysis, but is fortunately comparatively uncommon. It is due to injury to the brachial plexus. It is most frequent in breech-presentations, particularly where the arms are extended, but it may also occur in other difficult labours, such as a shoulder-presentation (by pulling on the arm), or a vertex-presentation (by axillary traction). It may be associated with fracture of the humerus or clavicle. Very rarely the lesion is bilateral.

The symptoms are characteristic. At first there may be some slight pain or tenderness about the shoulder. The muscles affected are the deltoid, biceps, brachialis anticus, and subscapularis. Quite exceptionally all the muscles are affected as the result of very extensive damage to the brachial plexus. The arm hangs in a characteristic attitude; it is flaccid, and the palm of the hand looks outward. Atrophy of the affected muscles follows, but is difficult to appreciate in a late infant. The diagnosis, as a rule, is easily made. From acute poliomyelitis the condition is distinguished by its congenital origin, and from a congenital hemiplegia an Erb's paralysis can be diagnosed by the facts that the arm is flaccid and the leg normal. Care must be taken to exclude the presence of a fracture of the humerus or clavicle, with which the traumatic palsy may be associated.

The prognosis is bad. Within the first three months of life considerable improvement may occur, but final complete recovery is exceptional. The deltoid, in whole or in part, is usually permanently affected. The prognosis should be guided by the electrical response of the affected muscles (p. 200).

The treatment at first consists in keeping the paralyzed arm warm, and in gentle massage of the affected muscles. Electrical massage should be tried if no improvement occurs in the first month. But it is with difficulty applied at such an early age. Where the deltoid alone remains paralyzed a nerve-suture operation may be undertaken later. The damaged portion of the fifth nerve is divided, and the peripheral part sewn into the sixth nerve, a form of treatment which has not with some success.

Birth-palsy of the Lower Limbs is a very rare event, but has been described as the result of injury to the vertebral column involving the spinal cord.

Cerebral Birth-palsies.—It is well known that neonatal hæmorrhage is a frequent cause of still-birth, or of death occurring in the course of the first few days of life. That such hæmorrhage should be survived, and the child grow up with cerebral diplegia, is another matter. This question is discussed on p. 474.

Hæmatoma of the Sternomastoid Muscle may be conveniently alluded to here. It is due to injury to the muscle during labour, and is most frequently found in first-born children and in cases of breech-presentation. It is very much more common on the right than on the left side of the neck. Rarely it is bilateral. It is due to hæmorrhage into, and partial rupture of, the muscle, and has nothing directly to do with inherited syphilis. A rounded swelling is seen at or below the middle of the muscle soon after birth. There is no discoloration. The swelling is very hard to the touch. The head is pulled down on the affected side, with the face turned to the opposite side. The blood is rapidly absorbed, and some fibrosis occurs at the site of the hæmorrhage. Usually, within three months the abnormality has entirely disappeared and the head is held normally; but occasionally such a hæmatoma produces a permanent torticollis, with which may be associated hemiatrophy of the face. On this account a guarded prognosis must be given. No treatment is necessary or advisable during the early stages.

CONGENITAL CEREBRAL PALSIES.

Most commonly a congenital cerebral paralysis is diplegic in type, less often paralytic, still less frequently hemiplegic, and very rarely interplegic. As a rough rule, it may be said that a diplegia is congenital in origin, while a hemiplegia is post-natal or acquired; but it is a mistake to adhere too rigidly to such a rule as this. Cerebral diplegia is in many instances an acquired condition, and infantile hemiplegia is occasionally congenital.

We may take, therefore, cerebral diplegia as our type of a congenital cerebral paralysis, the other forms differing only in the localization and extent of the lesion in the brain. It must be remembered, however, that in cerebral diplegia there may be cerebellar involvement, and that there are cases in which the cerebellum is the only abnormal part of the brain (p. 477).

CONGENITAL CEREBRAL DIPLEGIA.

Etiology.—The etiology of this condition has been the subject of much controversy, and the various theories advanced to account for it are briefly these: (1) Meningeal hemorrhage is well-known to occur during a difficult delivery, and accounts for as many as 50 per cent of still-births (Spencer); or it may cause death within a few days of delivery. The theory of the causation of cerebral diplegia which was the most widely held until recently was, that the condition was due to a meningeal hemorrhage occurring at birth, but not proving fatal. It was thought that various parts of the brain became compressed and atrophied by the extravasated blood. Pathological evidence is, however, directly against this theory, and it has now been discarded by nearly all anatomists. (2) In place of a gross hemorrhage it was thought that minute hemorrhages due to asphyxia during delivery might cause the death of the nerve-cells; or (3) That the deleterious effect of some toxin including syphilis might be the causative agent. Neither of these views, however, appears to explain the morbid changes usually found post mortem. (4) It is now generally held that the condition is not due in most instances to degenerative changes occurring in the brain, but to a non-development of the nerve-cells in various parts of the brain.

Clinically, there are one or two points of etiological significance which require mention. Both difficult and precipitate labour have been frequently noted in the births of these patients, and it was thought that they favoured the theory of the disease being due to meningeal hemorrhage; but they may equally well be explained by the assumption that they are due to failure of the uterine expulsive powers, and to feeble development of the fetus respectively. Again, such a child is often the first-born of a family; but in that case it is usually the only one born to elderly parents. More often cerebral diplegia is found in a child born late in a large family, when the reproductive powers are becoming exhausted.

Morbid Anatomy.—As a general rule a condition of atrophic sclerosis is found post mortem in cases of congenital cerebral diplegia. In certain areas the surface of the brain is depressed and here the gyri are much shrunken, hardened, and rather yellow in colour. The corresponding sulci are much broader than normal. The condition is sometimes called a "walnut brain." These abnormalities occur in various parts of the cerebrum, and may or may not be found in the cerebellum. When examined microscopically, the affected gyri are seen to consist almost entirely of neuroglia, and to contain no nerve cells. There may be small cysts in the substance of the gyri (pericystically). The blood-vessels are not the seat of any obvious disease.

Congenital cerebral palsy may be produced by other congenital malformations, such as absence of portions of the brain, cystic disease of the brain (the so-called "budding" brain), and vascular disease.

The pyramidal tracts are undeveloped, or imperfectly developed, not degenerated.

Symptomatology.—The symptoms in a case of congenital cerebral paralysis will vary with the localization and extent of the lesion in the brain.

The motor system is the one most affected. As has been mentioned, the spastic paralysis may be diplegic, paraplegic, hemiplegic, or even possibly monoplegic in type, and, further, may vary much in degree



FIG. 81.—CONGENITAL CEREBRAL DIPLEGIA.—Child being Propped Up.

as well as in distribution. The diplegic variety is the most common. The symptoms are usually first noticed when the child attempts to make use of its limbs. The local weakness is only very slight in most cases; the arms suffer more than the legs. Spasticity is more pronounced than the weakness, and contractures are very prone to develop. Perverse movements, clonic movements, athetosis, and tremor are common, and are probably due to involvement of the mid-brain. In the legs the spasticity is such that when the child is held up the feet

cross one another, owing to adductor spasm, and the child walks on tip-toe with the legs crossed (cross-legged progression, Fig. 84). The reflexes are those of spastic paralysis. Dysphagia may be present (infantile pseudo-bulbar paralysis). Blindness is rare.

In severe cases rigidity of the spine may be noted from birth, and opisthotonos is occasionally so marked as to cause a mistaken diagnosis of posterior basal meningitis. A condition of pleurothotonos in such a case, under the care of Dr. Sutherland, is shown in Fig. 85.



Fig. 85.—INFANT CLINICALLY SUSPECTED OF AN INFANTILE PLEUROTHOTONOS.

Mental development is commonly deficient owing to the affection of the prefrontal lobes. It is very varying in degree, from slight backwardness to profound dementia, and is not necessarily in any relationship to the amount of spastic paralysis. Thus, some cases come under supervision for their mental state, and only a careful examination shows that they belong to this group (spastic idocy). This can prove so devious. Speech is acquired late, and is apt to be indistinct and defective.

Cerebellar symptoms, which may be present, are considered later.

Diagnosis.—The severe cases of generalized rigidity in infants are to be differentiated from posterior basic meningitis by the absence of acute symptoms (convulsions, excepted) and of bulging of the anterior fontanelle, and by the normal state of the cerebrospinal fluid. Where the condition is of a less grade of severity, and where nothing wrong is noticed until the child should begin to walk, it may be difficult to be certain whether the case is congenital or acquired. As a rule, careful enquiry into the progress of the child's physical development, and into the illnesses that it has passed through, will settle the point. Congenital cerebral diplegia will make itself apparent at an earlier age than such a condition as Friedreich's ataxia, but ataxotonic family-idiocy in its later stages may possibly be mistaken for it. The hardness of the muscles of the spastic limbs may suggest pseudo-hypertrophic muscular atrophy.

Prognosis.—The severe infantile cases do not survive long, and die of marasmus or bronchopneumonia within a few months.

In the less severe cases the prognosis must be made on the extent of the paralysis and the amount of mental defect. Too gloomy a view should not be taken of the child's mental development, for with training much may be accomplished. In the most favourable cases the child may later be able to work under supervision at a simple trade. Epilepsy is prone to develop, but will often cease after a few years.

Treatment.—Of first importance is the education of the child's mind, for the greater the mental development the more the use that can be made of the affected limbs. For this reason, careful training should begin early. For the spasticity of the limbs, passive movements and educational methods for increasing the voluntary power and control, are of great value, and should be practised from the beginning. If contractures have not been prevented, tenotomies may be necessary.

CONGENITAL CEREBELLAR DIPLEGIA.

(*Congenital Cerebellar Ataxia*).

Etiology.—The separation of cerebellar from cerebral congenital diplegia is only a matter of clinical convenience and is not founded upon any essential pathological difference. Thus the condition is due to maldevelopment of some portions of the cerebellum. Part of the organ may be absent, but more commonly there are found in it areas of sclerosis exactly comparable to, and co-existent with, the cerebral sclerosis described above.

Symptomatology.—The symptoms of cerebellar diplegia consist of ataxia, hypotonia, nystagmus and scanning-speech. All or any of these may be present, and to these may be added symptoms of cerebral

involvement, of which the most constant is some degree of mental deficiency.

From experimental work (Luciani) it seems clear that absence or underdevelopment of portions of the cerebellum only gives rise to characteristic symptoms if the cerebrum is also to some extent involved, but such involvement may not be of sufficient degree to originate symptoms by which it can be recognized clinically.

Diagnosis.—Congenital cerebellar ataxia has to be distinguished from other cases of ataxia in children.

Ataxia in childhood is found in three groups of cases as described by Dr. F. E. Blatten:—(1) Congenital ataxia, the type we are considering here. (2) Acquired ataxia of sudden onset (acute ataxia), the type due in most cases to cerebellar encephalitis and described on p. 212. (3) Acquired ataxia of gradual onset and usually progressive in character. Friedreich's ataxia accounts for most cases in this group. Cerebellar tumours, usually tuberculous in children, require mention here. Disseminated sclerosis might produce a clinical picture resembling some of these cases, but its occurrence in children is very doubtful. Dr. Gordon Holmes has shown ("Brain," 1907) that there are no satisfactory grounds for continuing to describe Marie's hereditary cerebellar ataxia as a separate disease.

The diagnosis of the congenital group must therefore rest upon the history of the case. The ataxia should be observed as soon as definite purposeful movements are made and walking is attempted.

Prognosis.—A good deal of improvement may be expected to occur in the absence of signs of severe cerebral involvement.

Treatment.—Educational methods, both physical and mental, are to be adopted in order that the child may learn to make the best use of his powers.

2. FAMILIAL NERVOUS DISEASES.

We have here to consider certain nervous diseases which tend to occur in more than one member of a family. These must be regarded as "inborn" and thus require separation from ordinary acquired diseases; but inasmuch as no symptoms are present until a variable time after birth, they cannot be classed as congenital diseases. They may therefore be termed familial diseases, and we have here to deal with such of them as show symptoms during childhood.

FRIEDREICH'S ATAXIA.

The most common familial nervous disease of childhood is that described by Friedreich in 1861, and which bears his name. It is sometimes called hereditary ataxia.

Etiology.—No definite parental taint can be traced in this disease, and as single cases occurring in the middle of families are not very uncommon, it seems unlikely that disease or alcoholism in the parents is a factor of much importance. As a rule, more than one member of the family is affected. Both sexes appear about equally prone to the disease. Any acute illness may predispose towards the first appearance of symptoms.

Morbid Anatomy.—The spinal cord has been noted as being abnormally small in many cases. It shows widespread sclerosis, which affects chiefly the posterior columns, but in addition the lateral columns and direct cordellar tracts. Lissauer's tract usually remains unaffected, the cells of Clarke's column may be atrophied.

Symptomatology.—The symptoms usually appear about the sixth year of life. They may be later or earlier, but do not begin during infancy. As a rule, the first symptom is ataxia, and the first physical sign that of loss of knee-jerk; but there is considerable variation in the early manifestations of the disease, depending upon what tracts in the spinal cord are first affected.

The ataxia is first seen in the legs; the gait becomes clumsy and lurching; the feet are kept wide apart and are put heavily to the ground. At its worst, the ataxia prevents the patient walking or even standing. The movements of the arms become clumsily tremulous and ataxic, and the head and trunk swayingly. The faces is usually dull, but although emotionalism is common, actual mental deficiency is exceptional. Nystagmus is usually present, and scanning speech may develop. Certain deformities are common. Of these club-foot is the most important, as it is often recognizable early in the disease. *Mare au griffe* is less common. Scoliosis is usually present, but is a rather late development. The deep reflexes are as a rule absent early, and remain so throughout the disease. The plantar responses are extensor in type.

Bladder trouble, sensory or trophic changes, and optic atrophy have been described, but are exceptional.

As has been mentioned, the early signs of the disease depend upon what tracts in the spinal cord are first affected, and thus they are variable. It is important to remember that there is a definite group of these cases in which the knee-jerk is at first increased, and not lost until later.

Diagnosis.—Where only one member of a family is affected, the diagnosis may be of some difficulty. It may be mistaken for disseminated sclerosis. This, however, is in children a disease of the very greatest rarity, and indeed it ever occurs in them. Should the knee-jerk be increased, the similarity is very marked. The presence of deformities and the gradual downward progression of the disease

will distinguish Friedrich's ataxia. As a rule the deep reflexes are lost, and the disease simulates tabes and not disseminated sclerosis. From tabes it is distinguished by the absence of signs of inherited syphilis and Argyll-Robertson pupils and the presence of extensor plantar responses.

By the history of the age and mode of onset, Friedrich's ataxy may be differentiated from congenital cerebellar diplegia (p. 277) and cerebellar encephalitis (p. 214).

THE PERONEAL TYPE OF MUSCULAR ATROPHY.

This disease is also known under the names of the neuritic, or, since it was described almost simultaneously by three neurologists, the Charcot-Marie-Tooth type of muscular atrophy. From 1880, when it was first described, until quite recently, there has been much controversy as to whether it should be included under the myopathies or the neuropathies. Recent work, however, seems to have proved it to be due to degenerative changes in the spinal cord, and since it tends to run in families, it is described here as a familial nervous disease, and thus separated from myopathy which is a familial disease not dependent, so far as is known, upon any disease of the nervous system.

Etiology.—As in other familial diseases, the occurrence of isolated cases in a family is not infrequent. Both sexes may be affected. The transmission through the female line is less clearly marked than in some other hereditary conditions.

The first symptoms arise in young subjects, usually during the second half of childhood. Occasionally there follows upon some acute illness.

Morbid Anatomy.—In the spinal cord degenerative changes have been found in the anterior cornual cells, in the lateral and posterior columns, and in the peripheral nerves. The muscular atrophy is probably to be regarded as due to the changes in the anterior horns, which are most marked in the region of the lumbar and cervical enlargements. In the affected muscles there are the signs of a simple atrophy, with none of the changes characteristic of myopathy.

Symptomatology.—The wasting is first seen in the peroneal muscles, both legs being about equally affected. Thence the atrophic changes spread to the anterior tibial muscles and to some extent to those of the calves. This is the stage of the disease most commonly met with in children. The thighs are normal, the knee-jerks active; but below the knees the legs are severely wasted and the movements, especially those of dorsiflexion of the toes and feet, are very much impaired. Double talipes varus develops. Rarely, the disease is al-

first unilateral. The wasting soon spreads to the thighs, attacking first the vastus internus muscles (Fig. 87).

As the disease progresses the wasting becomes marked in the small muscles of the hands, and spreads to the forearm. Later, the muscles of the upper arms, and then those of the pelvis and shoulders, become atrophic. The facial muscles remain unaffected.

The atrophied muscles show, in addition to the wasting and loss of power, diminution or absence of response to tactile stimulation. Fibrillary tremors are frequent but not constant. Some impairment of sensibility is occasionally present, especially on the legs.

Course.—In its course the disease may show periods of quiescence. In some cases the arrest is permanent, but more often after a varying lapse of time, amounting sometimes to years, the disease once more begins to develop. On the other hand, it may progress from the start, uninterrupted by any remissions. Perhaps a stay in the development of the disease most often occurs at the stage where the legs alone are atrophied, or where the hands are also involved, but an arrest may occur at any time.



FIG. 87.—PERONEAL TYPE OF MUSCULAR ATROPHY.

Diagnosis.—With a family history and a typical distribution of the atrophy, such as is found in children, there is no difficulty in recognizing the condition. In older patients, where the changes are widespread there may be more doubt as to the diagnosis. From acute poliomyelitis it is distinguished by its slow onset and gradual progress. The age of the patient (in children), and the distribution of the wasting, differentiate it from progressive muscular atrophy. The atrophies show no fibrillary tremor in the affected muscles, nor does the wasting develop as in this disease. In them the upper arms and thighs are early affected, in peroneal atrophy the wasting shows first in the legs and hands. Toxic peripheral neuritis is not likely to lead to difficulty, as the forms occurring in children bear little resemblance to the disease we are considering.

Prognosis.—No cure can be expected. An arrest in the progress of the disease may occur, but is hardly likely to be permanent, although occasionally this may happen.

Treatment.—Massage and galvanism, together with the administration of strychnine in full doses, appear to have some influence in lessening or arresting the progress of the disease. In advanced stages restorants may be of service in enabling the patient to walk.

WERDNIG-HOFFMANN PARALYSIS.

An extremely rare affection, which tends to occur in several members of a family, is known by this name, after the two observers who first described it. It is also called "progressive spinal muscular atrophy of infants." The disease usually appears within six months of birth, and shows itself by a progressive flaccid and atrophic paralysis.

It starts in the legs, shoulders, neck and trunk, and spreads to the distal muscles, and ultimately involves those of deglutition and respiration. The affected muscles show fibrillary tremors. The disease runs a rapid course, varying from a few weeks to a few years. Usually death occurs before the end of the third year.

The disease is a myelopathy, the spinal cord showing the lesions of a progressive anterior poliomyelitis. From the simple atrophic type of myopathy occurring in infants (p. 504), this disease is distinguished with some difficulty. Fibrillary tremors, rapidity of course, severity of symptoms, and involvement of the muscles of deglutition and respiration would be in favour of the myelopathy.

Prognosis.—There is steady deterioration, and the patient becomes permanently crippled. Death usually occurs from some intercurrent affection before the fourth year of life is reached.

Treatment.—We cannot hope to arrest the disease. Attention to the general health by means of such drugs as iron, arsenic, and strychnine, and by hygienic methods, is of some value. Massage adds to the comfort of the patient. Tenotomy for the contractures are of very little use.

AMAUROTIC FAMILY IDIOCY.

This, sometimes known as Sachs's disease, or more correctly as Warren Tay's disease, is described on page 444.

HEREDITARY OPTIC ATROPHY.

(Leber's Atrophy.)

A very rare disease, sometimes occurring in several generations and in several members of one generation, and characterized by the rapid, chronic sufferer, onset of blindness. Males are chiefly affected, females rarely suffering, although they transmit the disease. Blindness as a rule comes on about the age of puberty, but may occur as early as the fifth year. It is bilateral and may be permanent. In

some instances improvement occurs. The ophthalmoscopic changes are those of slight optic neuritis, followed by atrophy. The origin of the disease, if indeed it be a definite entity, is unknown.

FAMILY SPASTIC PARALYSIS.

Certain rare cases have been grouped together under this name. They are characterized by occurring in several members of a family, the spasticity arising at about the age of puberty. It is thought that they are due to atrophic changes (posture decay) in the lateral columns of the cord. Occasionally atrophic paralysis is added, constituting a family type of amyotrophic lateral sclerosis. In neither type can treatment prevent the progress of the disease.

FAMILY PERIODIC PARALYSIS.

This disease, which is one of great rarity, is characterized by the onset of temporary flaccid palsy. It occurs in several generations and in several members of one family, and has been reported as being present as early as the second year of life. The paralysis may affect any of the muscles of the trunk or limbs, and as a rule lasts only for several hours or days. The onset of the paralysis is gradual and may be preceded by some sensations of numbness or tingling. A case has been carefully described by Singer, in *Brain* (1901).

3. ACQUIRED NERVOUS DISEASES.

ACQUIRED CEREBRAL PALSIES.

Elsewhere it has been stated that congenital cerebral palsies are usually of the paraplegic or diplegic type, while those of post-natal origin are generally of hemiplegic distribution. It was, however, pointed out that the histories of cases did not invariably enable a certain differentiation between the congenital and acquired groups, and that while some cases of diplegia were of the latter type, some hemiplegic palsies were congenital.

Acquired Cerebral Diplegia is for the most part due to gross diseases of the brain, such as meningitis or tumour, which produce symptoms of a more pressing nature than those of the spastic paralysis. A chronic syphilitic polyneuritis may, however, give rise to little trouble apart from the diplegic condition.

Acquired Hemiplegia.—The term "*infantile hemiplegia*" is much used, but it is nevertheless one not devoid of danger. Under it are included both congenital and acquired cases, and even those which occur not in infancy but in childhood. Nor does it refer only to one disease, but to the result of many conditions, some of which can be closely traced pathologically, while others are extremely obscure. It is well, therefore, to bear in mind that in labelling a case one of infantile hemiplegia, we are not making the diagnosis of a disease, but

of a symptom of several diseases. Where the origin of the paralysis is quite unexplained, we have no other course open to us than to speak of the condition as one of infantile hemiplegia.

We have, therefore, to consider to what causes, so far as they can be traced, hemiplegia in children may be due.

The congenital cases have already been considered (p. 473). They are probably due to atrophic sclerosis or other maldevelopments of part of the brain. During delivery a hemiplegia may result from meningeal hemorrhage, but probably such cases do not survive for more than a few days at the most. Asphyxia at birth may perhaps account for some cases, but this is doubtful.

Infantile hemiplegia is most commonly found in children under three years of age, and it is during infancy that cases arise which are the most difficult to explain. In many the onset of the hemiplegia synchronizes with an attack of convulsions, and in a certain number of these the convulsions are probably the actual cause of the paralysis by inducing a small hemorrhage in the brain. In others, the convulsions cannot be regarded as the origin of the hemiplegia but rather as a symptom of the onset of the condition. Thrombosis of a cerebral vessel during convalescence from any of the acute specific fevers appears an undoubted cause of certain cases. A small hemorrhage occurring during a paroxysm of whooping-cough is a rare cause. Acute poly-encephalitis (p. 274) certainly accounts for a few cases, but only for a small proportion. As Dr. Still has shown, if all cases of post-natal infantile hemiplegia are taken together, no definite seasonal incidence can be shown corresponding with that of poly-encephalitis and myelitis. Other cases arise with the same symptoms, but are probably not of the same nature. There are fever, convulsions, and hemiplegia, but the causes both of the constitutional disturbances and of the paralysis (whether inflammatory, thrombotic or hemorrhagic) are quite obscure.

Inherited syphilis accounts for a few cases, usually in children over three years of age. The lesion is probably due to thrombosis following syphilitic endarteritis. The thrombosis is of rapid onset and may be preceded by temporary warning attacks of paresis. Signs of inherited syphilis are usually present, and the eye-gonads particularly should be carefully examined for them. Intracranial tuberculosis may produce hemiplegia of sudden or of rapid but not instantaneous origin. The former is probably due to an embolus of caseous material. In the three well cases the author has seen, tuberculosis meningitis appeared two, four, and six weeks after the onset of the paralysis. In no case was a post mortem obtained. It is possible that such a condition might lead to a permanent hemiplegia without setting up a fatal meningitis. In the type with rapid but not sudden onset, the hemiplegia is associated with the earliest signs of tuberculous meningitis and is due to blockage of the vessels in one Sylvian fissure by the meningitis already present.

Emboliæ associated with heart disease (usually malignant endocarditis) is an occasional cause of hemiplegia in older children.

Prognosis.—As has been mentioned in dealing with the congenital cerebral plexes, the ultimate condition is one in which spasticity is as a rule more marked than the paralysis. Moreover, the face is only temporarily affected, and the recovery of the lower extremities is more complete than that of the upper.

Mention must be made of some obscure cases of infantile hemiplegia, in which the paralysis passes off completely in a few hours or days. In others so great an improvement occurs that the hemiplegia is scarcely noticeable after a few years.

Often, however, the paralysis is permanent, and improvement occurs only to a limited extent. With hemiplegia the mental disabament is not likely to be more than simple backwardness. Epileptiform seizures may occur.

Treatment.—The treatment is on the same lines as that given for congenital cerebral paralysis (p. 477). If any cause for the condition can be traced, such as infected sepiæ, this should be treated.

VENOUS SINUS THROMBOSIS

Etiology.—(1) Thrombosis of a sinus, usually the superior longitudinal sinus, may occur in malarial infants, particularly where there has been much diarrhoea. It is due to the extreme infestation of the circulation, and is termed malarial thrombosis. (2) As a complication of various infections, pneumonia, measles, and other acute specific fevers, sinus thrombosis may occur. (3) It may be part of a general pyæmia. (4) The sinus may be infected and thrombosed from direct extension from an extracranial abscess. (5) In a large group the infection of the sinus is by means of the veins opening into it, and is associated thus with middle-ear and mastoid disease, cellulitis of the face, suppuration in the nasopharynx, and disease of the scalp or cranial bones.

It is to be remembered, however, that a suppurative meningitis is more common than a sinus thrombosis as an intracranial complication of otitis media in children.

Symptomatology.—In the malarial cases the symptoms are in no way characteristic. High fever and head-extension may be present, but these may occur without any sinus thrombosis. The external veins of the forehead are rarely distended. The diagnosis of the condition can hardly be made, nor is it a matter of great moment, as the thrombosis must be regarded as a terminal condition rather than as the actual cause of death.

The symptoms of the secondary infective group of cases are those seen in older subjects: repeated rigors and an oscillating fever, with

cerebral symptoms. Without definite signs in the external veins the diagnosis between sinus thrombosis and intracranial abscess cannot be made, except by exploratory operation. Examination of the cerebrospinal fluid should prove negative in both these conditions, thus differentiating them from acute meningitis.

INTRACRANIAL ABSCESS

Excluding tuberculous cases, the first five or six years of life enjoy an almost complete immunity from intracranial abscess. Over half the cases are secondary to middle-ear and mastoid disease, the abscess arising in connection with the chronic rather than the acute types of disease. Middle-ear disease is associated particularly with cerebral abscess, mastoid disease with cerebellar abscess. The path which the infection takes is not as yet satisfactorily settled. Disease of the bones of the nasal cavity causes a small number of cases in children. Injury to the skull is the next most important cause of intracranial abscess. In addition there are some remote causes of intracranial abscess. Of these the most important is bronchiectasis, while empyema, pyæmia, and malignant endocarditis may also be mentioned.

Symptomatology.—The symptoms may be divided into acute and chronic.

The acute symptoms may be present from the beginning of the formation of the abscess, or may occur after the persistence of the chronic symptoms for an indefinite period. They strongly resemble those of acute meningitis, which may indeed be co-existent. Thus there are headache, cerebral irritation, vomiting, and some delirium. The temperature may be subnormal, raised, or intermittent. The pulse tends to be slow. Some rigidity of the neck may be present. Convulsions at first may be localized and are slight, later becoming general and severe. Some hemiplegic weakness may develop, together with optic neuritis. As the case proceeds, the convulsions become more severe, the fever higher and accompanied by sweating and thirst, and death occurs in coma within, as a rule, a week or ten days of the onset of the acute symptoms.

The chronic symptoms, when they are well-marked, resemble those of an intracranial tumour. They may, however, be very trifling or even absent in the latent form of the disease. Attacks of headache, vomiting, and slight fever, with possibly occasional convulsions, may occur. As the abscess grows, the signs of raised intracranial pressure become more marked and more persistent, optic neuritis develops, but localizing symptoms are usually absent, as the supratentorial abscess is so frequently the seat of the disease. Cerebration may be delayed and the pulse is usually slowed. The clinical picture, then, is one of an inoperable cerebral tumour. Paralysis of the third nerve of the same side as the abscess, with contralateral hemiplegia, may be

present. Cerebellar symptoms or word-blindness may develop. The blood shows a leucocytosis. Sudden death may occur as in tumour, from some unexplained cause, but more commonly acute symptoms such as have already been described arise before the fatal ending of the disease.

Diagnosis.—The diagnosis of intracranial abscess is one of great difficulty, and can only be suggested by the possibility of such a condition being kept constantly in mind. In all cases where there are signs or symptoms of raised intracranial pressure, special attention should be paid to the condition of the ears and to the history of past otitis media.

From acute suppurative meningitis abscess can often only be distinguished by the normal condition of the cerebrospinal fluid, but as has been mentioned, acute suppurative meningitis may co-exist with an abscess. An abscess may also be mistaken for tuberculous meningitis. Here, again, an examination of the cerebrospinal fluid will enable a differentiation to be made. It may in addition be a help to remember that abscess very rarely occurs under the age of six years, while tuberculous meningitis is less common at that age than at an earlier date.

From intracranial growth an abscess in a chronic stage can hardly be distinguished except by a history of signs of past middle-ear disease. A polymorphonuclear leucocytosis would be in favour of an abscess.

Prognosis.—Unless surgical interference is successful, there is no hope of saving the patient. Acute symptoms almost invariably arise sooner or later, and prove rapidly fatal. If the abscess is opened recovery may take place, but this is by no means certain. Acute meningitis or some thrombosis may also be present, and the injury to the brain caused by the abscess is as a rule severe. More than one abscess may be present.

Treatment.—The treatment of the condition is entirely surgical.

HYDROCEPHALUS.

The term hydrocephalus is used to denote internal hydrocephalus. In this there is an abnormal accumulation of fluid within the lateral ventricles.

External Hydrocephalus is a very rare condition, of pathological interest only. As the result of hæmorrhage or inflammation, and usually in association with some malformation of the brain, there is a collection of fluid outside the brain but within the dura mater.

Acute Hydrocephalus is a term which is not now much used. It formerly denoted acute meningitis, which was in the majority of cases tuberculous in origin, and is used at the present time in its synonymy with tuberculous meningitis.

Spurious Hydrocephalus is a term sometimes used to denote a

condition clinically resembling tuberculous meningitis (i.e., acute hydrocephalus), but due to no gross intracranial lesion. This group of symptoms, spoken of elsewhere in this book as "meningismus," may be found in many diseases—such as pneumonia, otitis media, acute diphtheria, typhoid fever, influenza, and others.

Chronic Internal Hydrocephalus. the condition meant when the word hydrocephalus alone is used, has here to be considered.

CHRONIC INTERNAL HYDROCEPHALUS.

Etiology.—*Congenital Hydrocephalus*.—This may start as a primary condition, being due to maldevelopment when it is frequently associated with spina bifida and hydromyelia. On the other hand, it may be secondary to some form of intra-uterine meningitis, possibly, but certainly not invariably, due to syphilis.

Acquired Hydrocephalus.—This is usually secondary, and its most common causes are two in number—posterior basilar meningitis and a basal intracranial tumor. Of these the former is the more frequent cause. It is to be remembered that the hydrocephalus is the result of the sclerosing meningitis following that active stage of infection; that the onset of hydrocephalus may be delayed for several weeks or even months, and possibly years; and that it may be due to a very slight or abortive attack of posterior basilar meningitis, for some cases of hydrocephalus, in which no clinical evidence of the infection is obtainable, are found on pathological examination to belong to this group.

The ordinary posterior basilar meningitis is due to the meningococcus, and has nothing to do with inherited syphilis, but it is said that certain cases of acquired hydrocephalus are due to a basal leptomeningitis due to syphilis. This is, however, doubtful (p. 156). Lastly, hydrocephalus may come on in later childhood. In most of these cases no cause can be traced.

In those cases where the origin of the hydrocephalus is known, the disease is spoken of as a secondary. In others as primary or idiopathic. Dr. Leonard Guthrie regards some of the latter as due to a simple hypersecretion of the choroid plexuses (*Practitioner*, 1910).

Pathogenesis.—The cerebrospinal fluid is secreted by the ependymal lining of the nervous canal, particularly, if not wholly, by the parts overlying the three pairs of choroid plexuses which are situated respectively in the floor of the lateral ventricles and at the roots of the third and fourth ventricles. It is absorbed by the vessels of the meninges (possibly chiefly by those of the lower part of the third spinalis), and probably to some extent by the vessels of the pachymen bodies. In passing from its seats of origin to those of its absorption, the fluid has to flow through certain communications between the ventricular cavities and the arachnoid space. Of these the most important are the fine canals at the region of the anterior perforated

spots and the three openings in the roof of the fourth ventricle, one of which is situated centrally (foramen of Magendie) and the other two laterally (foramina of Luschka).

Hydrocephalus may be produced by obstruction to the paths of exit of the cerebrospinal fluid or to an overproduction of the fluid. In many cases both these factors co-exist.

The foramina of Magendie and Luschka may be obliterated as the result of postnatal haem meningitis or by the pressure of a brain tumour. The canals at the anterior perforated spot may become much dilated as a compensatory change, but in many cases of meningitis these too are obstructed.

Over-production of cerebrospinal fluid is, for the most part at least, dependent upon congestion of the choroid plexuses. The blood from these plexuses passes along the veins of Galen (bring in the *vena cava superior*) into the straight sinus. As there is no anastomosis with the superficial cerebral veins, obstruction to the veins of Galen, or to the straight sinus (by pressure or constriction), produces congestion of the vessels of the choroid plexuses and an increased secretion of cerebrospinal fluid.

Morbid Anatomy.—In addition to the changes causing the hydrocephalus which may be present, we have to note the results which hydrocephalus produces on the brain. The surface of the cerebrum shows much flattening of the gyri. The lateral ventricles show the greatest enlargement of their cavities, the bodies being more affected than the cornua. The cortex is compressed and thinned, sometimes to an extraordinary extent. The choroid plexuses are often hyperplastic in chronic cases, while in the more acute ones they may be flattened and bloodless. The ependymal lining of the ventricles is thickened, roughened, and injected.

The third ventricle is usually less dilated than are the lateral ventricles. The aqueduct of Sylvius may be dilated or may be obstructed. The size of the fourth ventricle depends upon the patency of the aqueduct. The posterior part of the cerebellum and the medulla are pushed down into the foramen magnum, which as a rule prevents any of the excess of fluid passing into the arachnoid space of the spinal cord.

Symptomatology.—As a rule, enlargement of the head is present. Where hydrocephalus arises in infancy, the enlargement may become extreme. The anterior fontanelle is bulging and much widened, and the bones of the skullcap can be felt to be separated. The shape of the head is very characteristic. The forehead protrudes, so that the head is oval in shape. The parietal eminences are carried backwards, the vertex and occipital surfaces being flatter than the frontal surface. The external auditory meatus runs directly inwards or even slightly backwards, instead of being directed slightly forwards.

Large veins are seen coursing over the scalp. The eyes may be so depressed by bulging of the orbital plates that the pupils are covered by the lower lids, and it is remarkable that even in such an extreme case the child may have sufficient sense left to draw the lids downwards, so that he may see. Retraction of the upper lids, producing the cerebral or hydrocephalic stare, may be seen in slighter cases (Fig. 42, p. 100).

In great contrast to the enlarged head is the small wasted face, so that from the front the face seems triangular in shape.

Where the skull is fully ossified and unyielding, as in older children, little or no enlargement of the head can occur. Hydrocephalus may even co-exist with microcephaly.

Various nervous symptoms are associated with hydrocephalus. Their intensity is least where the skull is most yielding, and greatest where enlargement of the head cannot occur. They simulate the general nervous symptoms of an intracranial growth. Convulsions are common. Mental deterioration is the rule, but it is frequently very much less than the cranial enlargement might lead one to expect. The children are drowsy and cerebriation is slow. Spasms of the limbs is usually present and symmetrical. Optic neuritis, more frequently optic atrophy, primary or secondary, may be found. Headache is not as a rule a very severe symptom in young children. Occasional cerebral vomiting may occur. Any unilateral signs, with the exception of unilateral fits, are very rare. Cerebellar symptoms may be present.

In older children, where the skull is unyielding and the onset of the hydrocephalus rapid, these nervous symptoms are of great severity. Headache, usually paroxysmal, may be extremely severe, and is associated with vomiting, optic neuritis, and convulsions. The symptoms are those of an unlocalized growth. Death usually takes place rapidly.

In addition to the nervous symptoms, those of severe malnutrition usually develop.

Diagnosis.—The diagnosis is clear when there is great enlargement of the head, but, as a rule, impossible apart from this, unless there is some recognizable cause for the onset of hydrocephalus.

Difficulty may occur where there is only slight cranial enlargement present, when the hydrocephalic head has to be differentiated from the rachitic and microcephalic.

From rickets the diagnosis is made by the differences in the shape of the head. The rickety head is square, the forehead large, but upright; in hydrocephalus, the head is oval in shape, the forehead protruding and overhanging the line of the face. A bulging fontanelle, apart from swelling or convulsions being present at the time, means increased intracranial pressure, and thus hydrocephalus, acute or chronic. The auditory meatus is normal in direction in rickets. The

may occur in both hydrocephalus and rickets. Signs of bony rickets may be present in an infant with hydrocephalus.

Macrocephaly means a large head normal in shape, and is usually easily distinguishable.

In any case of doubt the diagnosis will become clear if the case be watched. If the head grows more than an inch in circumference per month, hydrocephalus is almost certainly present.

Prognosis.—In severe and in progressive cases the outlook is hopeless. In slight cases a spontaneous arrest may occur. There is likely, however, to be some degree of mental impairment, though this will not increase if the disease becomes arrested, unless epilepsy develop. Most infantile cases die before the fifth year.

Treatment.—In the rare syphilitic cases mercury and iodide may be given. In the majority of cases, however, no treatment is of avail. Lumbal puncture, repeated daily, is of use in incipient cases, but is only applicable to them. It usually gives temporary relief, but commonly, after several punctures have been done, it is found that no more fluid can be withdrawn: the obstruction, which was at first partial, has become complete. In rare instances, however, this does not occur, and a cure results. For chronic hydrocephalus many operations have been tried, but hitherto have met with very little success, and have been for the most part abandoned.

INTRACRANIAL TUMOURS.

Only such points will be dealt with here as are of interest in connection with intracranial tumours as they occur in children.

The most common of these tumours in childhood are those of tuberculous origin. In the post-mortem records of cases of intracranial tumour tuberculous masses are found in from one-half to three-fourths of the total number of cases. But such figures as these would give a greatly exaggerated idea of the clinical importance of tuberculous cases, for in a large proportion no signs pointing to intracranial tumour are present during life. It is not at all uncommon to discover unexpectedly a tuberculous mass, or perhaps more frequently masses, in an autopsy on a child who has died of a tuberculous meningitis of the ordinary type. Tuberculous tumours, however, do form an important clinical group in children, and in cerebellar cases are the most frequent kind of tumour found. Elsewhere, however, they are not so common as such types as glioma and sarcoma. Gliomata are extremely uncommon in children, and endotheliomata very rare. Gliomata in the brain secondary to neoplasms elsewhere practically never occur in children.

As regards the sites of intracranial tumours in children the cerebellum is the most frequently affected part of the brain. Any space, however, below the tentorium is a common place for a tumour,

while supratentorial tumours giving rise to clinical signs are extremely rare in childhood.

Certain difficulties in the diagnosis and localization of a growth are particularly marked in children. In the first place, localizing signs are apt to be less definite in children than in adults, for the various parts of the brain are not in childhood so specialized for their particular functions as they are later in life, and a functional compensation readily occurs to deal with any localized destruction of brain tissue. A large lesion is therefore necessary in a child than in an adult to produce the same degree of loss of function; and inasmuch as nearly all tumours in children are subtentorial, the onset of hydrocephalus is frequent and may cause death before localizing signs are developed.



FIG. 100. "CHERRY-LAKE TEST" IN BLOOD.

On the other hand, certain false localizing signs are peculiarly apt to be present. Unilateral convulsions may be of no localizing value, as they may occur merely as the result of the hydrocephalus present. From the same cause a double hemiplegia may rapidly develop. Localizing signs may also be concealed, owing to the impossibility of carrying out any tests which are at all delicate. A great functional exaggeration of some symptom due to organic disease may mask its true character. For instance, slight weakness of an arm due to an incipient hemiplegia may be exaggerated into a total flexor paralysis of the limb.

There is, however, one particularly helpful sign in some cases of

intracranial growth in children, namely, local bulging of the skull. This is best marked in cerebellar cases, and is present in the majority of this group. Examination of the surface of the skull should never be omitted in dealing with cases of intracranial tumour in children.

Cerebellar Tumours in children are almost invariably of the intracerebellar type, and as a rule give rise to typical signs and symptoms which need not here be detailed. Dr. Frederick Bates has described a peculiar tilt of the head (the cerebellar position) which is present in the majority of these cases. The head is inclined towards the shoulder on the side of the lesion, the face being turned to the opposite side with the chin slightly tilted upward (Fig. 88). Such a position does not, however, mean of necessity a cerebellar tumour, for it may be found in cases of tumour elsewhere in the brain (Fig. 89), in which the cerebellum is compressed.

The earliest symptoms of a cerebellar tumour are usually very indefinite; slight ataxia, with an occasional headache and sudden vomiting. It is to be particularly remembered that the early signs are not persistent, but tend to come and go. To exclude the possibility of a cerebellar tumour early in its growth is a matter, therefore, of great difficulty, and there is no little danger of regarding the case as functional in type.



FIG. 88.—Symptoms of Pons.
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Pontine Tumours may produce the ordinary picture of crossed hemiplegia with involvement of the fifth, sixth, and seventh nerves. A nuclear lesion of the sixth nerve is shown by loss of conjugate movement of the eyes towards the side of the lesion.

Hypertrophy of the Pons is a name sometimes given to a diffuse glioma of the pons. There is universal enlargement of the pons, and as its shape is maintained it looks like the pons of an adult attached to a child's brain (Fig. 89). Such a condition is not uncommon in children. The initial symptoms in these cases may be extremely indefinite, and are usually more cerebellar than pontine in type. Optic atrophy is not seen until late, and headache and vomiting are only occasional and slight. There may be a little indefinite and transient ataxia from time to time. The child appears dull and sallow, and may show various symptoms of a functional type. For several months nothing further may develop, for such growths progress slowly, and are still slower in producing destruction

of the parts in which the growth is present. Later, the child may show a rather characteristic myopathic facies, with slight denture proclia and little or no wrinkling of the forehead. Definite cranial nerve palsies, optic neuritis, and spasticity develop still later. The disease advances, often running a slow course of from a year to eighteen months, and death ultimately occurs.

SYRINGOMYELIA.

This disease is occasionally seen in children, and runs as a rule a progressive course. It is sometimes associated with a congenital dilatation of the central canal of the cord (hydromyelia), with which, however, the disease has no real connection. The pathology of the disease in children is the same as that seen in adults.

DISSEMINATED SCLEROSIS.

It is doubtful whether this disease occurs at all in children. If so it is both extreme rarity. Clinically, a picture very similar to disseminated sclerosis may be met with in children, due to cerebellar encephalitis (p. 214), or to an atypical form of Frobenius's ataxy (p. 478).

INFECTIONS OF THE NERVOUS SYSTEM.

These are for the most part dealt with elsewhere in this book.

Acute Meningitis.—Tuberculous and meningococcal meningitis are described on pages 121 and 104. Acute suppurative meningitis due to septic organisms other than the pneumococcus differs in no way from the pneumococcal form, and the reader is referred to the description on page 110. Influenzal meningitis has been mentioned on page 208.

Chronic Meningitis.—This is usually due to syphilis, and is described on pages 189 and 200, where juvenile tabes dorsalis and general paralysis are discussed.

Chorea.—This is described on pages 157 to 170.

Acute Pulio-encephalomyelitis.—This, and its two subdivisions, poliomyelitis and polio-encephalitis, are described on pages 200 to 222.

Landry's Paralysis, always a disease of great rarity, practically does not occur during the years of childhood. If the severer forms of acute poliomyelitis are not properly appreciated (p. 219), there is no little danger of making a mistaken diagnosis of Landry's paralysis. It is worth bearing in mind, therefore, that acute poliomyelitis is very common in children, while Landry's paralysis is almost unknown in them.

Peripheral Neuritis.—In the great majority of cases in children this is due to the diphtheritic poison (p. 241), while a few are the result of influenza (p. 209).

Occasionally cases are seen which are apparently of the nature of a multiple toxic neuritis, but which cannot as yet be classified with any of the known groups of this condition.

SECTION XI.

DISEASES OF BONES AND MUSCLES.

I.—DISEASES OF BONES.

ACHONDROPLASIA.

ACHONDROPLASIA, or chondrodystrophy, was formerly known under the term of "fetal rickets," owing to a mistaken conception of its pathogenesis.

Etiology.—From the third to the sixth months of fetal life there occurs in achondroplasia a cessation of ossification in the primary cartilages of the bones, while the bone-formation from membrane or periosteum is unaffected. As the result of this the long bones are very short and thick.

Very rarely the condition has been found to be hereditary. It is, however, exceptional for these patients to have any children.

Symptomatology.—Achondroplasia produces quite a characteristic appearance. There is considerable dwarfing, a height of not more than four feet being reached with full growth. The head is large, the vault of the skull being rather hydrocephalic in shape, owing probably to premature closure of the sutures at the base. In fact all achondroplasias are extremely alike. The forehead is large, the bridge of the nose is depressed, the nostrils are wide and the jaw is frequently rather overhanging. The teeth show no characteristic change. The palate may be narrowed or normal.

The limbs are peculiarly short, the deformity affecting especially the upper arms and thighs. The hands are very characteristic. They are smaller than normal, and the fingers when extended are separated from each other at their distal ends (ray-like hands). The middle and ring fingers are particularly wide apart, the latter appearing to be set on a level posterior to the others, and so being partly concealed by them from the palmar aspect (brow on wrist). All the fingers are much the same in length.

The trunk appears too long for the length of the body, but would be normal in size for a subject of greater height. Considerable lordosis is usually present owing to tilting of the pelvis. There is some bending of the limbs, the knees in particular having the appearance of

being dislocated. The gait is waddling. The arms as they hang are not closely approximated to the sides of the body, and do not reach below the level of the great trochanters of the femora. Muscular development is good. The sexual organs may remain of the infantile type, but this is not the rule in these cases.

Mentally, achondroplasia generally occupies a level below the normal, and was occasionally weak-minded. They are usually, however, sharp and shrewd, able to read and write. The "volatile" Miss Mowbray



FIG. 10.—ACHONDROPLASIA (Age 2 1/2 years).



FIG. 11.—ACHONDROPLASIA (Age 3 years).

or "Dival Copperfield," was clearly an achondroplase, while the medical contributors were frequently dwarfs of this type. It is interesting to note that certain of the early Egyptian gods are figured as achondroplases.

Diagnosis.—Achondroplasia has to be distinguished from rickets, hydrocephalus, and cretinism. The characteristic shortness of the limbs is, however, sufficiently striking to enable it to be easily differentiated. It may be well to remember that in the normal

infant the umbilicus is midway between the top of the head and the soles of the feet: in achondroplasia this mid-point falls considerably above the level of the navel owing to the shortness of the legs.

Prognosis.—Many achondroplasies die at birth or during infancy. Throughout childhood they are apt to be frail and delicate, but after this period is successfully passed the condition has no influence on shortening life.

Treatment.—Achondroplasia cannot be modified by treatment.

CLEIDOCRANIAL DYSOSTOSIS.

(*Diastrophania*.)

This is a rare condition, in which there is defective ossification of certain of the membrane-bones. A large deficiency is left in the ossification of the vertex of the skull, the bones failing to approach each other at the high bend of the anterior fontanelle (Fig. 23). This is distinguished from the so-called fontanelle of hydrocephalus in that it is congenital, very extensive and associated with an enlargement of the head.

The clavicles are also affected their outer parts either being absent or the represented by fibrous tissue only. Owing to this abnormality the shoulders are so freely movable that they can be made to meet under the chin.

The condition may be both hereditary and familial. All the recorded cases (39 in number) have been collected by Mr. D. C. L. Fitzwilliams (*Lancet* 1910).

Many of these children are weaker and die during infancy; but should they survive childhood the deformity is not of great moment to them. The ossification of the skullcap is usually complete by the twentieth year: it rarely remains permanently deficient. The abnormal condition of the clavicles, on the other hand, is life-long. No treatment of the condition is at any avail.



FIG. 23.—CLEIDOCRANIAL DYSOSTOSIS. BOWEN-CLAR.

OSTEOGENESIS IMPERFECTA.*(Fragilitas Ossium; Infantile Osteomalacia.)*

Etiology.—Osteogenesis imperfecta is a rare disease of which the cause is unknown. It is not due to syphilis, and has probably nothing to do with osteomalacia of the type that occurs in adults, and which is very rare before the age of puberty and unknown before the fifth year of life. Nor is it a congenital form of rickets, although countless instances of the disease have been described as "congenital rickets." In some cases it is hereditary (15 per cent) and familial.

The cause of the disease, whatever it may be, acts during intra-uterine life, and renders ossification of the shafts of the bones and of the bones of the cranial vault defective. Since it is the periosteal ossification which is at fault, Dr. Canby has suggested calling the condition "periosteal aplasia." The bones affected are extraordinarily fragile and porous, and break on the very slightest provocation or even spontaneously.

Symptomatology.—The excessive fragility of the bones and their tendency to fracture may arise during intra-uterine life and cause the death of the fetus, or may result in injuries during birth which may prove immediately or rapidly fatal. Only a very few of such cases survive. In a few instances the tendency to fractures is not seen at all events in any marked degree, until the child is two or three months old. The disease exists in varying degrees of severity.

Commonly, the most careful handling will bring about fractures. These are practically painless and unobservable. In some cases spontaneous fractures occur. In these some tenderness and swelling arise at some spot in the shaft of a bone, and after a day or two a fracture is found to have developed at this site. In addition to being abnormally brittle, the affected bones are abnormally soft, so that they are prone to bend as well as to break.

The shafts of the long bones of the limbs are most affected, but the bones of the hands, the clavicles, ribs, and even the mandible, may suffer. The bones of the vault of the cranium are not always affected, but should they be, shortening of the antero-posterior diameter of the skull to a grotesque degree may result.

The fractures in most instances unite well, but with the formation of an excess of callus. Failure of union is rare.

Diagnosis.—The picture of the disease in its usual form is quite unique, and can hardly be confused with any other condition. Osteomalacia of the adult type, as mentioned above, does not occur in infancy. Rickets may be excluded by the age of the patient, the excessive fragility of the bones, and the normal condition of the epiphyses. Nodules of callus on the ribs at the sites of past fractures might be mistaken for a rickety nurse.

Prognosis.—Many of these cases are born dead, or die during infancy. Should the patient survive, the excessive fragility of the bones passes off after two or three years and no further fractures occur. Great deformity, however, remains.

Treatment.—During infancy the chief object of treatment is to attend to the general nutrition of the patient. Little can be done to avoid fractures or deformities. The fractured limbs may be bound up in wool and a soft bandage, but in some cases even this is impossible. Any attempt at the use of splints is as a rule quite impracticable. Later, should the child live, surgical treatment for the resulting deformities will be necessary.

OXYCEPHALY.

Oxycephaly, or "tower-head," is a rare condition, probably dependent upon some faulty development of the skull. Occasionally it is found in more than one member of the same generation of a family.



Fig. 93.—OXYCEPHALY.

The characteristic shape of the head, pointed in front and rising so to dome posteriorly, is seen in Figs. 93 and 94. The eyes are usually

markedly proptosed, and often show optic atrophy. Mental development may be up to, or rather below, normal.

Other malformations have been noted in many cases, of these



Fig. 10. Craniotomy.

this most common are maldevelopment of the elbows, whereby full extension of the forearms is prevented, and various abnormalities of the digits.

II.—DISEASES OF MUSCLES.

MYOPATHY.

Myopathy or muscular dystrophy is to be regarded as a disease in which, independent of any changes in the nervous system, muscular atrophies occur. In the various types of myopathy, not only are the muscles the primary seat of disease, but the histological changes found in them are broadly similar.

The conception, therefore, of myopathy as a disease is founded upon a pathological basis, while its division into various types or groups of cases is merely a matter of clinical convenience.

It is important at the outset to grasp the fact that the different types of myopathy are in no sense different diseases, but are simply different manifestations of the same disease. This is shown not only by the similarity in the pathological changes in all the types, but by the occurrence in myopathic families of instances of more than one type of the disease, and by the occasional but well-known occurrence of cases intermediate or transitional between the ordinary clinical forms of the disease.

The differentiation of myopathy into clinical groups is, however, very convenient, and is to a large extent founded upon the age at onset of the symptoms, the presence or absence of apparent hypertrophy, and the distribution of the muscular affection.

Etiology.—While the pathology of myopathy is becoming well recognized, its etiology is still extremely obscure. It is in many cases both a hereditary and a familial disease, but the occurrence of an isolated case in a family is so common as to be in no way remarkable. The changes in the muscles seem to be due to a degenerative process rather than to an arrested development only, although some embryonic factor may possibly be present.

Morbid Anatomy.—Microscopically the affected muscles show characteristic changes. The fibres in transverse section are for the most part much smaller than normal, and their angles are rounded off, so that in this view they appear round or oval in shape. Their nuclei are increased in number. A small proportion of the fibres, however, are greatly increased in size, and in cross section appear large, round, faintly striated, and often vacuolated. In longitudinal sections it is seen that cross striation is preserved to a remarkable degree, while longitudinal fibres are common. There are, in addition to these changes, collections of small round cells, an increase of connective tissue and fat, and thickening of the walls of many of the blood-vessels.

Changes in the nervous system may be entirely absent. Where present they are to be regarded as secondary in nature.

Symptomatology.—A few general points may be mentioned here before the symptoms of the various clinical types are described.

The mental condition in myopathy may be normal or below the average. Perhaps most certainly the patient is somewhat backward owing to the interference which the disease causes in his education rather than to any actual mental deficiency. In other cases, however, there seems no doubt that the mind is fully developed.

The affected muscles do not correspond in their grouping with any spinal cord lesion, nor do they show fibrillary tremor or reaction of degeneration. To both faradism and galvanism they give much diminished contractions, very strong stimuli being necessary to elicit any response. The patient feels pain with galvanic currents, but even

to strong irritants he is curiously insensitive. This is also seen in some cases of acute poliomyelitis, and in the infantile progressive muscular atrophy of spinal origin described by Wernig and Hollmann (p. 482). Sensation for touch, heat, cold, and pain are otherwise perfect. The muscles of the eyes, larynx, and pharynx are not involved. The deep reflexes are normal, diminished or absent, according to the condition of the muscle which is concerned in the response.

Clinical Varieties.—Emphasis has already been laid upon the fact that these are only different manifestations of one disease, so that intermediate cases, those which do not conform exactly to any one type, are to be expected.

The only type which is at all common in childhood is the pseudo-hypertrophic. Nearly all the other varieties, however, may occur in



FIG. 12.—MUSCULAR PSEUDOHYPERTROPHIC TYPE.

children, and thus require mention. The myotonia atrophica type has not yet been recorded at an earlier age than fifteen years, and so no further reference to it need be made.

The Pseudo-hypertrophic Type.—This rarely occurs in children under five or over ten years of age, and starts most frequently in those of five or six. It is almost exclusively confined to the male sex. Although it frequently occurs sporadically, it is often both familial and hereditary, transmission being through the unaffected female.

The earliest symptoms are those connected with the child's power of walking. It may be noticed that he becomes limberly, walks with a waddling gait, is prone to fall, and is unable to get up from the floor without assistance. Attention may first be drawn to the child's weakness by the great difficulty which he begins to show in ascending stairs, although able to descend with ease. The enlargement of the

calves is present at this stage, but is safely regarded by the parents as connected with the child's lack of power.

As the child stands, the enlargement of the calves and buttocks is very noticeable, and on closer inspection the infraspinati and deltoid muscles are found to be similarly changed. The thighs and upper arms are smaller than normal, and the folds of the axillae are seen to be much wasted. The last point is of diagnostic importance. Owing to it the outline of the thorax is unduly clear, and when an attempt is made to lift the child off his feet by putting the hands under his arms, a peculiar sensation of his slipping through the hands is experienced.

As the result of weakness of the muscles of the back lordosis develops, and the child stands with head erect, shoulders thrown back, and abdomen protruding. The gait is waddling, the trunk being moved from side to side in order that the legs may be swung forward.

The child endeavours to mount stairs in a very characteristic way. One leg is swung out laterally, and so placed upon the stair above. The hand is then placed upon the knee of that leg, and the weight of the trunk is thrown forward on to the hand. By straightening the knee, an action helped by the hand, the step is mounted.

A very regular set of movements is performed when the child gets up from the floor. If on his back he rolls over on to his face, and gets up on his hands and knees. By partially straightening the knees he gets on to his hands and feet. Then the feet are gradually approximated to the hands until they can clutch at the ankles. The hands are then by degrees brought higher up the lower limbs, grasping the shins, knees, and thighs in turn, and with complete straightening of the knees the trunk is raised to the vertical position.

When fully developed, pseudo-hypertrophy is found in the gastrocnemii, glutei, infraspinati, deltoid, and triceps muscles, while the wasting of the muscles of the thigh and of the biceps and axillary folds becomes marked. The most constant features of the type are perhaps the smallness of the folds of the axilla, and the enlargement of the infraspinati muscles. The muscles, whether small or enlarged, are very weak. In the later stages contractures develop, and the intercostal muscles are weakened. As a rule death occurs from some disease of the respiratory system.

Diagnosis.—In childhood the condition is as a rule easily recognized. In some cases of cerebral diplegia the spastic muscles are hard to the touch, and may appear enlarged; but here the condition has existed since birth, the deep reflexes are much increased, and the plantar responses are of the extensor type. The method adopted by myopathia children in getting up from the floor may be closely simulated by some cases of tuberculous spinal curvæ. Where contractures have developed, and the child is bedridden and the wasting of the muscles is extreme, the myopathy has to be distinguished from an old and severe case of acute poliomyelitis. This may be done by the history of a gradual onset and of the progressive nature of the malady, while

smallness of the axillary folds is strongly in favour of myopathy as opposed to a myelopathy.

Idiopathic Muscular Atrophy is the name given to cases in which the distribution of the muscular weakness is similar to that of the pseudo-hypertrophic type, but is not associated with any apparent enlargement of any of the muscles. The onset of these cases is usually during young adult life.

The Facio-scapulo-humeral Type.—This often known as the Landouzy-Dejerine type, and formerly as the infantile type, requires mention among the diseases of children inasmuch as the facial affection is either congenital or appears soon after birth. The muscles of the face which are involved are the orbicularis palpebrarum and the orbicularis oris, so that the eyes cannot be completely closed and the lips cannot be properly moved. In infancy suckling is interfered with. The face in later life is very expressionless, the mouth is large and imperfectly closed. The movements of the eyes, tongue, and jaw are normal. No further symptoms develop as a rule before the second decade of life, when wasting appears in the muscles of the shoulder-girdle, upper arms, and thighs. Both sexes are alike affected.

Diagnosis.—The diagnosis of the condition in infancy, in the absence of a family history of myopathy, is one of some difficulty, but its possibility should be remembered in cases of supposed double facial palsy, a rare condition which is most commonly due to double middle-cerebral disease.

Erö's Juvenile Type.—This closely resembles the Landouzy-Dejerine form, except that the face is not affected. It appears in either sex, usually during the second decade of life, and affects first the muscles of the shoulder-girdle. Occasionally pseudo-hypertrophy is apparent in some of the affected muscles. Later the upper arms, thighs, and buttocks become wasted.

The Distal Type.—This type, described by Gowers, Spiller, and Batten, is hardly as yet clearly defined. In it weakness and atrophy of the distal muscles are found, while the proximal muscles remain unaffected. It occasionally occurs in childhood. The clinical aspect of this type resembles closely that of the proximal type of muscular atrophy (p. 486), and differentiation between the two conditions is not always possible. Dr. Batten regards facial involvement as strongly in favour of the distal type of myopathy, and attention of sensation in the peripheral parts of the limbs as pointing towards the proximal type of muscular atrophy.

Under this heading may be mentioned on clinical grounds the hereditary type of atrophy of the hand-muscles described by Dr. Thompson as occurring in seven members of a family of five generations.

Myotonia Congenita: Simple Atrophic Type.—The pathological evidence proves conclusively that the condition known as myotonia congenita, or congenital myotonia, myotonia, and myoplasia, and as Oppenheim's paralysis, is a type of myopathy. There is much

evidence pointing to the fact that the cases described by Dr. Batten as the simple atrophic type of myopathy are the same as amyotonia congenita. Such cases as these were originally described under the name of the infantile type, but, as this is sometimes used for the Landouzy-Déjerine group, it is now discarded for the type under consideration.

The symptoms are either present at birth or develop in early infancy. The muscles of the limbs are small, and lack both power and tone, but show no localized atrophy or hypertrophy. All movements are feeble, but none are impossible. Most characteristic is the extreme flaccidity of the muscles. So marked is this that the hands and feet can be bent backward until their dorsal surfaces touch the limbs. The hands and feet are very long and thin. The mental condition of these children is normal. As a rule they never learn to walk, but adopt some other method of getting about, such as rolling or buttock-walking. From the peculiar squatting attitude which the child may adopt, Dr. Head has invented the term "frog-child."



Fig. 54.—Amyotonia Congenita.



Fig. 55.—Amyotonia Congenita.

Most of the subjects of this condition die during infancy. Should the child survive, some improvement may be looked for; possibly he will be able to walk with assistance. The deep reflexes have been reported as obtaining. In most instances, however, there is progressive deterioration and not proliferation. In late stages, contractures develop.

The histological changes in the nerves are similar to

those found in the other types of myopathy. Either sex may be affected. Amyotonia congenita has now been recorded as occurring

in myopathic families, but no such connection is usually to be traced.

In *diagnosis* myotonia congenita has to be distinguished from rocky hypotonia (p. 68), a condition in which the histological muscular changes do not resemble those of myopathy.

Intermediate Cases.—It is not necessary to deal with such cases as do not fit accurately into any of the forms of myopathy described above. As a rule a slightly irregular case is sufficiently like some recognized type to allow of its inclusion therein. The most common irregularities are perhaps the focal involvement of the latissimus. Definite type appearing in the pseudo-hypertrophic and other forms, and the presence of pseudo-hypertrophy in the Erb's juvenile type.

Treatment of Myopathy.—Beyond massage, the use of electricity, and the administration of strychnine in large doses, little can be done for myopathy. The progress of the disease may be lessened by these measures, and is occasionally arrested. Complete recovery has been recorded in unshakable cases, but is infinitely rare.

The child should be encouraged to take as much exercise as is possible, and should not be kept in bed until he is entirely bedridden. In the later stages tenotomies may be of some service.

MYOSITIS OSSIFICANS.

The initial stages of this rare disease are seen during childhood, and may present a very puzzling picture to one unfamiliar with the early signs of the condition.

Swellings appear in the various parts of the body, usually most marked on the back or in the neck. They are large, round, or oval bosses, and their development is not accompanied by any constitutional disturbance. They are not tender, and at the most cause only very little discomfort. Usually they appear in the muscles, are definitely circumscribed, and the skin over them is movable. Less often they seem to be more superficial and to be attached to the skin. In some cases they may show fusion before they disappear. They are not symmetrically arranged and very rarely pit on pressure.

These tumours may develop as early as the fifth month of life. At first they disappear in the course of a few weeks and leave no obvious traces; but later, perhaps after an interval of many months, fresh swellings arise, and although they subside to a great extent, they leave indurated areas in the muscles. These form the basis of the disease.

A very peculiar associated sign in myositis ossificans is the abnormal condition of the big toes which is frequently present. This is congenital (present at birth) and symmetrical. Most commonly the big toes are very markedly shortened and curved in under the second toes, but various other abnormalities have been noted.

As the disease progresses the diagnosis becomes clearly established by the terrible rigidity which is the result of the bony deposits in the muscles. The transient nature of the early symptoms is the point necessary to emphasize here, and at first no suspicion of myositis ossificans would cross the mind of one unfamiliar with the initial stages of the disease.

The aetiology is entirely bad. We know nothing of arrest or rise in this disease, either spontaneous or as the result of treatment.

APPENDIX A.

DIETETIC AND THERAPEUTIC MEASURES.

1. DIETETIC MEASURES.

Whey.—Two parts of milk warmed to blood heat (100° F.) add a teaspoonful of Fiechtil's Pepsinaria, Benger's or Lanesby's Benger. Mix, and allow to stand until the curd separates. Strain. The whey must be heated to 130°–135° in order to destroy the ferment, which will otherwise curdle the milk to which the whey is added. At 160° the lactalbumin coagulates, but should this occur it is no practical drawback.

Prepared thus, whey contains the lactalbumin, sugar, and sales of milk (p. 10), but only from about 1 per cent to 1½ per cent of fat.

The percentage of fat may be diminished by using skimmed milk for the preparation of the whey, or may be increased by thoroughly drying the curd with a fork before straining. In the latter case, when a full-cream milk is used, whey will contain about 1 per cent of fat.

The cost of whey prepared thus is 21 or 2½ pence per pint above the cost of the milk.

Whey, as purchased from a dairy, is quite clear, and contains practically no fat. It costs about 7½ pence per pint.

White Wine Whey.—Add 2½ oz. of cooking sherry to half a pint of warm milk. Bring to the boil, and allow to stand for three minutes. Strain through a double layer of butter muslin. This whey contains a fair proportion of fat and, in addition, an amount of alcohol equivalent to 25½ minims of brandy to the ounce (50½ minims). It has a stimulant and carminative effect, but is suited for temporary use only.

Tartaric Whey (Still and Hyem).—Dissolve 8 grains of tartaric acid in half a drachm of water. Add this to half a pint of milk just brought to the boil. Mix well, and allow to simmer for five minutes. Strain as before.

This whey is rich in fat, and costs only $\frac{1}{2}$ of a penny per half pint above the cost of the milk.

Barley Water.—From Pearl Barley.—Wash one tablespoonful of pearl barley, and put it in a saucepan with one pint of cold water. Let it come to the boil, and then simmer beside the fire for half an hour. Strain and use as required. Should be prepared twice daily. Thus prepared, barley water contains from 5 to 2½ per cent of starch.

From Prepared Barley.—A heaped teaspoonful of prepared barley is made into a paste with cold water. Add boiling water to the pint. Boil for five minutes, stirring constantly. Thus prepared, barley water contains 1–2 per cent of starch.

Lime Water.—Add 1½ gr. crystals of liquid calcic saccharine for every 5 oz. of milk mixture.

If liquid calcic be used, sixteen times as much (1-1 oz.) must be added.

Citrated Milk.—A solution of sodium citrate in water is prepared of such a strength that each drachm contains sufficient citrate for one feed, allowing 2 grains of citrate for each ounce of milk. Thus, where 5 oz. of milk are being given in each feed the solution should contain 5 grains of sodium citrate to the drachm.

To citrate the milk, add a drachm of the solution to the feed, and bring the mixture up to the feed, as in the usual process of scalding milk for use. This is preferably done in the infant's bottle. Allow to cool until suitable for feeding.

Where more than one feed is prepared at a time the milk may be allowed to cool and be warmed again for use.

Barrogh's and Wellcome's powders, consisting of two grains of sodium citrate. If used they should first be dissolved in water as above.

In passing from citrated to unsaturated milk the amount of citrate added is gradually reduced.

Peptonized Milk.—Furchild's Peptogenic Milk Powder may be used. One-cupful (cup supplied) is sufficient to peptonize a pint of milk. No sugar need be added.

Warm the milk (or milk mixture) at the edge of the fire or by standing the vessel containing it in hot water. Add the powder, stirring constantly, and keep the milk at a temperature not exceeding 140° F. for a quarter of an hour. Then bring to the boil to put an end to the process. It is now ready for use.

For "half" peptonization the process may be prolonged to thirty minutes (or even longer), but in this a bitter taste is imparted to the milk. This develops after peptonization has proceeded between fifteen and twenty minutes.

In the case of older children peptonized milk may be flavoured with a little chocolate or coffee.

In passing from peptonized to unpeptonized milk the period of peptonization may be gradually reduced while the amount of powder is lessened, the capital being made up with lactose.

If liquid peptonization is used in grs. of sodium bicarbonate to the pint of milk must be added. The addition of sugar will also be necessary.



FIG. 100.—APPROXIMATE MEASUREMENTS OF HUMANIZED MILK. (Continued.) The stopper is used for preparing the milk.

Humanized Milk.—Prepare ½ pint of whey (p. 501), and mix cold with ½ pint of fresh milk. Add cream and sugar according to the strength required.

Where cream can be reasonably procured the whey and cream may be prepared thus: Take ½ pint of fresh milk, allow it to stand for twelve hours. Skim off the cream, and prepare the whey from this skimmed milk. The cream is added to the final mixture. This being gravity cream will contain only half the amount of fat that is present in bought or commercial cream. (8 per cent.)

Humanized milk should be prepared twice daily.

Humanized milk of varying strengths is prepared by several dishes (p. 144 V. 17).

Decalcified Milk.—Make the required quantity into a thin paste with water; add warm water (previously boiled) to the necessary strength, usually 1 to 10 or 1 to 2 (p. 45).

Albumen Water.—Place the white of a fresh egg in a cup, beat it slightly with a fork, so as to break it. Mix it well with half a pint of cold water. This may be done by shaking in a closed bottle. Lactose and a pinch of salt may be added. Afterwater tepid.

Albumen water may be made double this strength if required. 3 eggs, 3 oz. of lactose, and a pinch of salt to a pint of water.

Raw Meat Juice.—Take a quarter-pound of best raw mutton-steak (lean only). Shred it by scraping with a knife. Place in a vessel, add one ounce of cold water, cover with muslin, and leave for four hours in a cool place. Then transfer to a mortar (if oblong) and pound it well. Squeeze through muslin. If properly made this should provide 2 oz. of raw meat juice. Prepare twice daily.

In the case of older children this preparation may be administered in a red wine-glass or in some beef-tea or soup.

Raw Meat Pulp.—Shred the meat by scraping with a knife as above, or by passing the meat through a fine wire sieve. The pulp may be given in bread sandwiches.

Potato Cream.—Boil a potato and scrape away the part immediately under the skin. Beat this up into a thick mass with milk. Give 1-2 drachms twice daily.

2.—THERAPEUTIC MEASURES.

Gastric Lavage. Take about two feet of rubber tubing. Fasten one end to a glass funnel, and the other, through the medium of a piece of glass tubing, to a Jaquet's catheter No. 12. Prepare a solution of sodium bicarbonate, 3 drachms to 1 pint of water, and use at 100° F. Place a basin on the floor by the side of the patient.

Wrap the child up so as to insulate the arms. Lubricate the Jaquet's catheter with glycerin; pass it empty. In a small baby it is not necessary to do more than gently to help the involuntary expulsive movements; the tube is very easily passed.

Push the tube close to the mouth, fill the funnel with the fluid and hold it up so that the air in the tube down to the codonum bubbles up through the fluid. Now lower the funnel slightly, and keep it at such a height that the fluid flows gently into the stomach. When no more runs in lower the funnel, and allow the washings to escape into the basin. Repeat this until the washings are clear.

Push the tube again near the mouth and withdraw it gently.

Rectal Lavage.—This is done on much the same plan. A No. 7 or 8 Jaquet's catheter may be used. It is lubricated with oil and passed till

into the nostrils. Normal saline at 100° - 105° (double if these or much fever) may be used. The infant's buttocks rest on a surgical tray.

Nasal Feeds.—The same type of apparatus may be used as in gastric lavage, but it is not necessary to have so long a tube. Use a No. 3 Jaeger's catheter, and lubricate it with olive oil (glycerin is too irritating). Withdraw the tube quickly when the feed has been given.

Esophageal Feeds.—A No. 12 Jaeger's catheter, lubricated with sterine, may be used, as in gastric lavage.

Mustard Bath.—Prepare a bath of water as hot as can be comfortably borne by the elbow (110° - 105° F.). Take a quantity of mustard, one tablespoonful for each gallon of water, and make it into a thin paste with water. Add this to the bath. (Note that if powdered mustard be added dry to the water in the bath, some may float on the surface, and may burn the patient's skin.) Immerse the infant, supporting the head and shoulders. Add hot water as necessary to keep up the temperature of the bath.

When the infant's pulse has improved, and no cry becomes stronger (1 to 2 minutes), dry it rapidly with hot towels, and put it to bed in warm blankets and with hot-water bottles. A hot-water bed should be used if possible.

Subcutaneous Infusion.—Take four feet of rubber-tube tubing. To one end connect a hollow needle; to the other, a funnel (the funnel of a glass syringe answers well), which may be surrounded by cotton-wool in order to keep the fluid warm, and tied to the head of the cot.

Prepare a solution of normal saline at a temperature of 100° - 105° F. With a tube fast fast hang this and be delivered at the right height.

The sides of the chest are most suitable for these injections; the skin of the abdominal wall should be avoided. Prepare the skin surgically, and see that the rest of the body is warmly covered.

Fill the funnel with the solution, making sure that it is not below 140° . Allow some to flow through so as to warm the tubing. The assistant now pinches the tube (as a clamp may be used), and the needle (half of an inch), is pushed well in under the skin. All exposed parts are then covered by hot wool, and the fluid is allowed to run in under the skin, the funnel being refilled from time to time.

When between 1 and 3 oz. have flowed in the needle is withdrawn, and the skin wound covered with a small collodion dressing.

The infusion may be repeated every four hours if necessary.

Antitoxic Injections.—In making these it is advisable to make use of a small length of rubber tubing to connect the needle and syringe. By this means accidents from any struggling on the part of the child may be avoided. The abdominal wall should not be used for the injections; the axilla are preferable.

DRUGS

Administration of Drugs.—It is not always an easy matter to make a child take the medicine prescribed for it. More especially is this difficulty seen in connection with the first few doses. Many children will accept a medicine they have been compelled to take. Some children will always vomit after a powder, but this is quite exceptional.

Foodsters should usually be made up with some white sugar, and some milk may be given to wash the medicine down. It is not as a rule a good plan to suspend medicine in milk, lest the child should refuse afterwards to take milk again.

For infants the best flavouring agent is glyceria, which imparts a sweet taste to the medicine.

Children will sometimes take medicine better out of a medicine glass than out of a cup, sometimes vice versa.

Dosage.—The dosage of drugs in the case of children is not seldom a matter of unnecessary anxiety to practitioners.

The dose for most drugs may very easily be calculated by means of a single formula. For a child the usual adult dose may be divided by the fraction $\frac{\text{Age}}{\text{Age} + 12}$. Thus for a child of six years the dose would be $\frac{6}{18}$ or $\frac{1}{3}$ of the usual adult dose.

By this means a safe dose may be easily selected, and there is small need to limit the number of drugs to be used. For this reason no series of prescriptions is given here, for such tends towards such a limitation. The drugs may be selected freely for children as in adults, the only added consideration being concerned with their taste. In the instances of the most powerful drugs, however, such small quantities are necessary that the taste of them is of comparatively small moment. In the foregoing chapters of this book some of the most commonly prescribed medicines have been given.

Children show peculiarities with a few drugs. Some they tolerate remarkably well, e.g. belladonna, strychnine and chloral. Others they do not tolerate well, but special caution need only be used in the case of opium and morphine, and this practically only when dealing with infants.

Opium.—Infants, especially if in a state of collapse, are very susceptible to opium. For a general rule, during the first year of life, the fraction of opium may be given in the following doses: at a quarter of a year ($\frac{1}{4}$ month) 1 minim; at 6 months, $\frac{1}{2}$ minim; and at 1 year, 1 minim; and these doses may be repeated six-hourly. Tinct. camph. being only $\frac{1}{10}$ th of the strength of tinct. opii, may be given in larger doses.

In giving opium to children, especially to infants, it is often convenient to have the opium made up separately from the rest of the mixture, so that this drug may be given or withheld at any particular time. It is useless to give a sleeping infant opium.

Morphine, given hypodermically, should rarely be administered except to older children. It is convenient to have the B.P. injection (5 per cent) made up with an equal volume of distilled water, so as to reduce its strength to 2½ per cent.

Strichnia, for hypodermic injection, is conveniently made in quarter-mergits, in order that the smaller doses may be far more accurately measured. At a 1 in 100 solution a baby may be given 2-minim doses hypodermically every four hours as a last dose, but this cannot as a rule be kept up for more than twenty-four hours before slight violence and twitching movements are observed.

Infusion of Senna Pods.—As this is such a useful aperient preparation it may be convenient to give methods by which it may be prepared.

For Home Preparation.—For a child of 1 year. Take 5 pods, and pour

upon them 2 drachms of boiling water. Set aside for four hours, and at the end of that time pour off the 2 drachms of fluid. The first dose may consist of two drachms, to be followed nightly by half doses. For a child of 5 years, 4 or 5 pods may be used.

On a Large Scale.—The infusion of senna pods may be conveniently prepared by the method devised by Mr. Reynon, Dispenser at the Paddington Green Children's Hospital. A quantity of pods is infused in the ordinary B.P. method, starting with boiling water, for not less than four hours. The infusion is poured off, and then percolated twice through the pods. Water is added to the infusion, so as to make it a 1 or 2 solution, i.e. 4 lb. of pods make 4 or 8 fluid oz. of the finished product. A little amount of chloroform is added as a preservative. Infants may be given $\frac{1}{2}$ —1 drachm, older children 2 drachms at night.

APPENDIX B.

SOCIETIES, INSTITUTIONS, ETC., AIDING
INVALID CHILDREN.*

It not infrequently happens that the doctor in dealing with the sick children of poor parents finds that his patients cannot be properly looked after at home, or that they require special nursing, food, instruments or convalescent treatment which the parents are quite unable to supply. Under these circumstances there are many authorities which may be called upon to render such assistance as is needed.

Inasmuch as the patient may suffer if the methods of obtaining assistance are not understood, it is proposed here to set forth very briefly some of the ways in which help may be obtained for the sick children of the poor.

1.—GENERAL AND PROLONGED AID AND SUPERVISION.

The Invalid Children's Aid Association.—This is the only society which will help and supervise, under the doctor, the treatment of a child for a number of years, and in most cases where aid is needed, it is usual to apply at once to this association.

It excludes new cases over the age of 14 years, and such as by means of their after-destination are regarded as better left to the authorities of the Poor Law. Incurable or hopeless cases it will assist towards obtaining help from other sources.

The society is closely connected with the Charity Organisation Society, and the cases are dealt with on much the same lines. Pending investigation, temporary assistance is procured. All cases are dealt with in accordance with the advice of the doctor in charge of the patient, and the society does not work without the cooperation of a doctor.

Uses.—Excluding such cases as have been mentioned, the I. V. A. A. may be used to aid all others. Not only will it see that the treatment ordered by the doctor is properly carried out, but it will assist the parents in obtaining instruments, special nursing or convalescent treatment for the child. Further, a matter of great utility to the practitioner, it will draft the case to any society suitable for any special help that may be required, thus saving the doctor much time and many difficulties. In this way it will deal with many problems that may confront the practitioner, and it is usually best to put the case straight into the hands of this association.

Admission.—It is only necessary that the doctor should send a note to the secretary at the head-quarters of the society, or to any of its local offices, or the offices of societies affiliated to it, certifying that his patient is in need of some specified help. The name will then be visited and investigations

* The author is much indebted to Miss E. A. B. Wilson, Lady Almoner in St. Mary's Hospital, for her kind assistance in revising this Appendix.

Note.—As a rule where the expenditure of money is required the parents are expected to contribute according to their means.

Address.—Denison House, Vauxhall Bridge Road, London, S.W. Applications may also be made to various local or provincial offices of the F. C. A. A. or of the Charity Organisation Society, Central Aid Society, Guild of Help, and kindred societies, which work in conjunction with the F. C. A. A. These are to be found in most of the towns in England and Wales.

II.—POOR-LAW CASES

For all Poor-law cases application must be made to the Relieving Officer of the district, either for the attendance of the parish doctor or for admission to the infirmary.

III.—BLIND CHILDREN.

The education of all blind children between 5 and 10 years is compulsory, the duty falling upon the local Education Authorities. These cases usually come under the notice of the Attendance Officers of the local schools and are reported to the Education Authorities by them, and the patients are drafted to a special school for the blind.

In cases of difficulty application should be made to the Invalid Children's Aid Association (p. 325), or, for patients in the Metropolitan area and adjacent counties, to the Union of Institutions, Societies and Agencies for the Blind, Denison House, Vauxhall Bridge Road, London, S.W.

IV.—DEAF AND DEAF-BLIND CHILDREN.

The arrangements for these children are similar to those for the Blind.

In cases of difficulty application should be made to the Invalid Children's Aid Association, or, for patients within the Metropolitan area, to the Royal Association in Aid of the Deaf and Dumb, 110, Oxford Street, London, W.

V.—EDUCATION OF PHYSICALLY-DEFECTIVE CHILDREN.

In London and other large towns special schools are provided for such children as are the subjects of chronic, non-infectious diseases. In them the school hours are shortened, the children are provided with a good meal in the middle of the day, and they are taken to and from school under the care of a nurse.

Admission.—Periodical examinations are held by the medical officer of the school upon whose opinion the child is admitted or refused admission.

Practitioners may facilitate the admission of their patients to these special schools by certifying that they are not fit to attend the ordinary school and are suitable for the special school. The names are then enrolled for examination at the next visit of the doctor in charge of the school.

The waiting-lists are often long, and there is need for increased accommodation at these schools.

VI.—EDUCATION OF DOUBLE-MIXED (MENTALLY DEFECTIVE) CHILDREN.

Under this heading are included on the one hand imbeciles and idiots, and on the other, children that are merely dull or backward. The group will therefore contain such cases as epileptics, congenital brain disease, chronic chorea, and others in which there is a condition of double-mindedness.

Education of such children is compulsory between the ages of 5 and 16.

The Education Authorities are bound to provide education for these children, and special schools or special classes have been set up for their reception.

Admission.—The Attendance Officer of the district should be informed. The practitioner may facilitate admission by certifying that the child is fit to enter for the ordinary school, and suitable for the school for mentally-defective children. The admission of the child ultimately rests upon the report of the medical officer of the school.

The Invalid Children's Aid Association (p. 515), or the National Association for Promoting the Welfare of the Feeble-minded, Devon House, 109, Vauxhall Bridge Road, London, S.W. may be consulted in cases of difficulty or doubt.

VII.—INSTITUTIONAL CARE FOR IMBECILES AND IDIOT CHILDREN.

Pauper Patients.—Application should be made by the parents to the Relieving Officer of the district. The practitioner may certify the mental state of the patient.

From the Metropolitan Poor-Law area the children are placed in the Burreth Asylum, Dartford, Kent. A few cases are taken at the age of 15 years, but in most cases admission is not until the 5th year.

Outside the Metropolitan area cases may be sent by the Board of Guardians to various County institutions or to asylums. In cases of difficulty application may be made to the Invalid Children's Aid Association for help.

Non-pauper Patients.—Admission to various institutions may be obtained by election or payment. The rules governing the eligibility of cases vary for the different institutions.

The chief institutions are:—

Earlswood Asylum, Earlswood, Redditch.

Eastern Counties Asylum for Imbecile, Imbeciles and the Feeble-minded, Colchester.

Royal Albert Asylum, Lancaster.

Western Counties Asylum, Starcross, Exeter.

Scottish National Institution for the Education of Imbecile Children, Larbert, N.B.

Stewart Institution for Imbecile and Imbecile Children and Hospital for Mental Diseases, Palmerston, Chichester, Co. Dublin.

VIII.—THE CARE OF CRIPPLES.

The medical condition of children who can be classed as cripples is so varied that there is little use in grouping them together.

It is evident that different forms of treatment may be required in different cases, e.g., instruments, active surgical treatment, prolonged rest or treatment with which education should be combined, convalescent treatment, etc.

Under these circumstances, where outside aid is required, it is usually best to invoke the help of the Invalid Children's Aid Association, the practitioner certifying what treatment would in his opinion be of most benefit to his patient.

IX.—HOME NURSING.

Nursing Limitations.—The assistance of nurses may be obtained for the sick poor at their own homes by application to the various centres from

which nurses are supplied. For the most part such nurses do not undertake the care of ordinary cases, but will undertake massage, preparation for operation, surgical dressings, etc.

For the Metropolitan area, the Queen Victoria's Jubilee Institute for Nurses, 28, Victoria Street, London, S.W. supplies without payment nurses for these purposes. The Institute has many local centres, and several other associations working on the same lines are affiliated to it. Application for information concerning its work may be made to the head office.

Some neighbourhoods also undertake the nursing of the sick in their own homes in various localities.

It is often wisest to apply to the Invalid Children's Aid Association (p. 512), who will pass the case on to the suitable centre for the supply of a nurse.

Individ Nurse.—Application for the services of a *district nurse* may be made to the Vicar of the parish, to a local district committee, or to the nurse herself, the arrangements varying in different localities.

N—CONVALESCENT TREATMENT

There are now so many convalescent homes, both seaside and inland, for children, that it is impossible to be familiar with the working rules of more than a few of them.

Under these circumstances it is advisable for the practitioner to ask the assistance of the Invalid Children's Aid Association, who will arrange where the child may be sent and what amount of the expense is to be borne by the parents. The doctor should give particulars as to the age of the child, the nature of the disease, the kind of climate required, and the length of stay advised.

In some cases letters for convalescent homes may be obtained from religious institutions or subscribers.

INDEX

	1884		1888
ARTHRICULAR disorders, con-		Adenoids, complicating whoop-	
— enlarged, dactylic aspect	260	— hoarse cough	246, 250
— examination in acute rheumatism	255	— etiology and symptoms	126
— motion in rheumatism	257	— indications for operation for	126
— pain, chronic spinal examina-	257	— laryngitis associated with	157
— tion in	258	— predisposing to asthma	159
— purpura	244	— nasal catarrh	121
— referred to children	23	— in dactylic lymphadenitis	129
— — chronic	151	Adenoid peritonsitis	129
— lymphitis in paranasal	143	Adenoid, carcinoma of cortex	426
— — paranasal	144	— — medulla	426
— — paranasal	96	— — hyperostosis	425
— — paranasal	145	— — hyperostosis	426
— — diagnosis of infection from	185	— — carcinoma	424
— — circulating typhoid	164	Adenoid, hemorrhages into	421
— — treatment	142	Adult, diseases of compared with	1
Atrophies, of breast milk	28	— children's	1
Atrophia, intermaxillary	258	Eggs, as evidence	155
— — intermaxillary fluid in	16	Age-incidence of acid dyspepsia of	121, 122
— — peritonsillar (acute)	147	— acute lymphatic leukemia	447
— — paranasal	129	— — poly-splenopharyngitis	211
— — pulmonary	115	— — primary psoriasis	492
— — diagnosis of empyema from	164	— — unsymmetrical family short	441
— — rhinopharyngeal	115	— — appendicitis	194
Atrophies, dental groups	82	— atonia	157
— — symptomatology	82	— children's disease	5
— — in tuberculous peritonsitis	111	— chronic	155
Atrophies	261	— — conservative treatment, paranasal	145
— — bone for	85	— — carcinoma	421
Atrophia	165	— — dipththeria	215
Atrophic rhinitis	115	— — pharyngeal orthostomatia, postin-	204
Atrophic rhinitis	115	— — gita	204
— — appearance of hands in	2	— — post-epidemic myopathy	161
— — acid dyspepsia	274	— — post-epidemic humeral myopathy	504
— — acute associated with	208	— — Pindborg's anemia	7, 678
— — — diagnosis from pharynx	165	— — heart disease, paranasal (167)	169
		— — rheumatic	268
		— — hypertrophic pharyngeal stenosis	7, 265, 272
		— — atrophic rhinitis, atrophy	504
		— — epithelial keratin	161
		— — epithelial atrophy	466
		— — consumption	7, 502
		— — general pharynx	161
		— — tubes	166
		— — laryngeal atrophy	116, 416
		— — liver atrophy	72
		— — lymphadenoma	417
		— — tongue	251
		— — laryngeal atrophy	306
		— — cubic tumors	466
		— — carcinoma	11, 195
		— — personal rhinitis, atrophy	466

Atresia due to intracranial tumours	493	Blood, diseases of	494
Pneumonia	475	— enlargement of spleen in	432
— agnathous of	470	— pressure, effect of subglottic on	155
— in infected syphilis	490	— syphilis of	155
— varieties of, in childhood	478	Breathing exercises in croup	106
Atrophic symptoms of diphtheria	491	Body-rocking	455
Atelectasis, acquired	340	Biting, prolonged, of soft, hard	
pulmonary congenital	492	— changes from	42
Atriochy, acute yellow, of liver	504	Bites, changes in nerves	38
— of lung, measles, bacterial	504	Bites, diseases of	495
Aural symptoms of adenoids	420	— and joints, syphilis of	182
Aspiration, method of	5	Bites, changes in rickets	64
		— leadings to adrenal carcinoma	514
B		Bites for artificial feeding	38
Bacillus	43	Brachial plexus injuries, paralysis	
Bacillus	502	— from	472
Bacillus coli in ecology of caecum	505	Brachycephaly	558
— pythia	491	Brain, coordination of parts of	14
— procyonius and acute gastritis	471	— irritability and epilepsy	464
— tuberculous mode of infection		Breast feeding	27
by	472	— digestive disorders in	36
Bacteriology of acute gastritis	471	— management of	45
— poliomyelitis	474	— atrophic fat	46
— diphtheria	491	— of syphilitic children	161
— epidemic diarrhoea	490	— milk, abnormalities of	38
— diphtheria	491	Breath-holding in croup	101
— whooping-cough	448	Breathing abnormalities due to	
Bart's disease	413	— adenoids	328
Bacterium in digest of milk	51	— with retropharyngeal abscess	513
— preparation of	506	— in tuberculosis meningitis	114
Bath, standard, method of preparing	511	— grouped in diphtheria	16
Baths in acute poliomyelitis	444	Bright's disease	595
Befudonia in measles	495	— syphilis	187
— rash resembling scarlatina	218	Bromide in diphtheria	190
— in whooping-cough	216	Bromides in the treatment of	
Berg's food, percentage	100	— spasms	461
— position of	54	Bronchial abscesses in rickets	68
Bercow's butyric acid in production		— complications of whooping-	
of acetone	81	— cough	712, 516
Bicarbonates of soda in digestion of		— glands, intervention of	146
milk	18	Brachycephaly	552
Bile, hypersecretion in		Bronchitis, acute	343
rheumatic arthritis	174	— etiology and symptomatology	342
Bile, congenital dilatation		— treatment	345
of	413	— associated with asthma	358
Biliary calculi	340	— chronic	353
Bile, attacks	340	— diagnosis of consecutive post-	
Bile, pain	472	— rickets from	348
Bladder, diseases of	497	— dilatation of stomach in	574
— stone in	498	— nephritis following	354
Blind children, avoiding radiation	228	— fibrosis	352
Blood, of the nose	322	Broncho-pneumonia, acute, tuber-	
Blind children, education of	510	culous	431
Blood, in myocarditis	514	— conservative strategy	345
— bacteria have invaginate	510	— prognosis and treatment	340
— anemia	510	— symptomatology	340
Blood changes in congenital heart-		— varieties, tropical and diag-	
diseases	187	— nosa	348
— pneumonia	51	— dangers in bronchitis	345
— in children	474	— diagnosis from atelectasis	181
— used in diagnosis of croup	194	— diphtheria	346, 343
— lymphatic proctitis	498	Bronchus, foreign body in	358
— pneumonia and tuberculosis	57	Brodie's method of feeding, &c., vol.	510
— primary tuberculosis	475	Bulbar crises of post-syphilitic	
— desquamated arthritis	472	paralysis	341, 345
— von Jaksch's anemia	445	— poliomyelitis	499

- Bullous pyoderma, congenital 186
 — varicella 452
 Burns in acute rheumatism 152
 Buttes, addition to roof of 37
- C**
 CACIOLI, Isidoro 129
 — real 467
 — vesical 466
 Calmette's test in pulmonary tuberculosis 134
 Calomet 225
 Calot's test 227
 Capillary keratosis 343
 Carcinoma, uterine, cervical
 — adnexal metastatic 423
 — of lobes 423
 Cardiac auscultation, congenital
 — changes in thorax 164
 — complications of diphtheria 242
 — disease (see "Heart") 537
 — dulness, etc., examination by 33
 — — in pericarditis 167, 168
 — measures in bronchitis and con-
 sistence leucocytopenia 530
 — rheumatism 151, 162
 — — influence of salicylates on 134
 — rhythm, disorders of 338
 — sounds of murmurs 235, 236
 — symptoms of acute septal 234
 — following measles 113
 — — in pericardial pneumonia 50
 — — pneumonia, treatment of 59
 Cardiovascular system, syphilis of
 Caries, dental 218
 Caries teeth causing symptoms
 stipulating groups 214
 Carriek's food, pericardial con-
 position of 33
 Carpal tunnel disease 418
 Carous lung, diagnosis of (symp-
 toms from) 164
 Carter, acute nasal 331
 — associated with acute bronchitis 343
 — chronic nasal 324
 Catarrh induced by reflux 85, 86
 Catarrh, gastric 223
 — laryngeal 345
 — laryngitis, acute 333
 — — diagnosis of diphtheria from 246
 — stomatitis 210
 — symptoms of adenoids 218
 — — as transitory stomati-
 tis 347
 — measles 232
 Catarrh, acute nasal in ticks 56
 Catarrh by extension of per-
 foration in eustachian 24
 Cerebellar diplegia, congenital 477
 — cerebellum 214
 — tonsils 327
 Cerebro-vertebrobasal system, re-
 cephalitis of 413
 Cerebral birth palsy 473
 — larynx 10
 — in tuberculous meningitis 331
 Cerebral diplegia 473
 — — diagnosis of chronic form 467
 — disease as cause of convulsions 453
 — hemiplegia in whooping-cough 247
 — — paresis, acquired 453
 — — congenital 423
 — symptoms of parainfluenza pneumonia 79
 Cerebrospinal fever, epidemic 208
 — R.S.L. importance in diagnosis 33
 — — in acute poliomyelitis 422, 423
 — — changes in thorax 166
 — — in diagnosis of intracranial disease 165
 — — — pneumococcal meningitis 112
 — — — toxic media 123
 — — — pneumonia 57
 — — pathogenesis of hydrocephalus 266
 — — post-biotic meningitis 166
 — — infectious meningitis 114
 Cestode 368
 Chapman's whole flour, percentage
 composition of 33
 Charcot-Marie-Tooth type of foot
 splat atrophy 436
 Chelone Maffei food, percentage
 composition of 33
 Chole, abnormalities in configuration 8
 — deformities due to internally 126
 — — as rachyosia 354
 — — following whooping-cough 245
 — — in ticks 54
 Chondral puncture in diagnosis
 of syngnath 262
 Chylo-Serous Swelling 16
 Chylous, diagnosis and treat-
 ment 253
 — etiology and symptoms of 253
 Children's diseases (compared with
 adults) 1
 Chiral in thorax 256
 Chloroform poisoning, delayed 82
 Chlorosis 415
 Chlorosis 415
 Chlorotic anemia in rheumatism 349
 — anemias 415
 Chlorosis 415
 — (syngnathic) 415
 Chondrochondrolysis 445
 Cholera infantum 285
 Chorea, cerebrovascular fluid in
 — acute 184
 — chronic 445
 — diagnosis of infant-spasm form 446
 — diagnosis and prognosis 187
 — and epilepsy 189
 — etiology of 187
 — headache as a cause, sign of 447
 — latent 245, 447
 — — symptoms in 186
 — — possible symptoms of 447
 — loss of speech in 168
 — partial paralysis and symptoms
 etiology 169
 treatment 163

- Diphtheria, *Corynebacterium* . . . 412
 — treatment of . . . 441
 Diplopia, acquired vertical . . . 481
 — congenital cerebellar . . . 472
 — cerebral . . . 474
 Diplococcus of meningitis . . . 464
 Dis-easing . . . 464
 Diseases of the bladder . . . 465
 — blood . . . 474
 — bones and muscles . . . 475
 — children and adults compared . . . 4
 — circulatory system . . . 464
 — digestive system . . . 465
 — genito-urinary system . . . 464
 — glands . . . 444
 — intestines . . . 475
 — kidneys . . . 464
 — larynx . . . 467
 — liver . . . 464
 — lungs . . . 464
 — Meckel's diverticulum . . . 465
 — nervous system . . . 462
 — nose . . . 467
 — oesophagus . . . 464
 — peritoneum . . . 460
 — pharynx . . . 465
 — skin . . . 464
 — respiratory system . . . 464
 — salivary glands . . . 465
 — sperm, blood, and glands . . . 464
 — sinuses . . . 465
 — stomach . . . 465
 Dislocation . . . 464
 Displacement of liver . . . 462
 Disseminated sclerosis . . . 464
 — — diagnosis of Friedreich's
 ataxia from . . . 470
 Distal myopathies . . . 464
 Diverticulum, Meckel's, diseases of . . . 465
 Dropsy of chronic nephritis . . . 462
 Drivens as symptoms in pneu-
 monia . . . 464
 — tuberculous meningitis . . . 462
 Drugs, administration of . . . 462
 — dosage of . . . 462
 — in nursing mothers . . . 462
 Dwarfism due to achondroplasia . . . 465
 Dyslexia . . . 466
 Dysentery, bacillary . . . 467
 Dyspepsia, acid . . . 471
 Dyspepsia in diagnosis . . . 46
 Dysuria . . . 465
 E All affections by tubercle bac-
 teria . . . 464
 — disease, diagnosis of pneumonia
 from . . . 46
 — symptoms of adenoids . . . 464
 Ear, examination of . . . 44
 Eclampsia eclam . . . 465
 Eclamptic and epileptic idiosy-
 crasy . . . 465
 Edema, and asthma . . . 465
 Education of blind, deaf, and dumb
 children . . . 460
 — in cases of chronic liver disease . . . 460
 — chronic . . . 460
 Education in insanity . . . 460
 — of bottle-nursed children . . . 460
 — the mentally deficient . . . 460, 461
 — physically defective children . . . 460
 Eileasms, diagnosis of serous and
 purulent . . . 465
 Electrical massage in stenoma-
 talgia . . . 472, 473
 — reactions in tetany . . . 445
 — in symptoms of acute poly-
 myelitis . . . 460
 Electricity in treatment of acute
 poliomyelitis . . . 461
 Embolism in etiology of tetraplegia . . . 464
 Emotional types of children . . . 445
 Empyema . . . 460, 461
 — empyemata, septic, and
 pyogenic . . . 460
 — diagnosis . . . 460
 — of serous effusion from . . . 465
 — peritonitis from . . . 445
 — etiology of . . . 460
 — general appearance in . . . 4
 — intrathoracic . . . 461
 — symptomatology . . . 460
 — treatment . . . 460
 Emphysema . . . 464
 — complicating whooping-cough . . . 447
 Encephalitis (see also "Polio-en-
 cephalomyelitis") . . . 464
 — cerebral . . . 464
 — of cerebro-spinal system . . . 465
 — cerebrospinal fluid in . . . 46
 Enteritis, erythritic, common
 hemiplegia . . . 465, 466
 Endocarditis, acute (rheumatic) . . . 464
 — etiology of . . . 467
 — fatal . . . 462
 — maligant . . . 460
 — — simulating typhoid . . . 464
 — — splenic in . . . 462
 — pneumococcal . . . 464
 — rheumatic, splenic in . . . 462
 — infective . . . 460
 Endothelium, intracranial . . . 464
 Enlargement of the thymus . . . 465
 Enteric fever (see "Typhoid") . . . 461
 Enteritis, pneumococcal in etiology
 of . . . 465
 — tuberculous . . . 461
 — — transition to diarrhoea . . . 465
 Enteron, culture in etiology of . . . 461
 — etiology . . . 460
 — prognosis and treatment . . . 465
 — as symptoms of systemic . . . 460
 Epidemic cerebrospinal fever . . . 460
 — diarrhoea . . . 464
 Erythematous, tuberculous . . . 464
 Erythema pain in rheumatism . . . 467
 Epilepsy . . . 461
 — and chorea . . . 464
 — convulsions due to . . . 467
 — following whooping-cough . . . 445
 — relationship of slight tetanus to . . . 460
 Epileptic and eclamptic idiosy-
 crasy . . . 445
 Epiphysitis, syphilitic . . . 462

- Epiphyseal epiphysis, age-related . . . 100
 Epistaxis . . . 514
 Erb's palsy, congenital . . . 504
 — paralytic . . . 472
 — from acute poliomyelitis . . . 205
 Ergot, in chloro . . . 110
 Eruptions of acute infectious fevers
 dates of rise also separate
 headings . . . 225
 — congenital syphilis . . . 179
 Erythema accompanying jaundice
 — neonatal . . . 474
 — rheumatic . . . 152
 Erythema, differential diagnosis
 of in scarletina, measles, and
 rubella . . . 138
 Eustachian tube, lesion in tuberculous
 tonsillar glands . . . 120
 Examination of children . . . 1
 — methods of . . . 5
 Exanthems in pneumonia . . . 98
 Extracranial abscess, entry through
 bone due to . . . 155
 Eye complications of measles . . . 111
 — eruptions in acute diarrhoea . . . 432
 — acute syphilis . . . 430
 — adrenal sclerosis . . . 424
 — syphilis of choroid vessels . . . 431
 — tuberculous meningitis . . . 423
 Eyelids, edema of . . . 424
 — swelling of, in scarlet . . . 77
FACIAL instability . . . 451
 — movements in chorea . . . 562
 — scarletina, bilateral . . . 504
 — birth palsy . . . 474
 — in cerebral diplegia . . . 475
 — septic parotitis a cause of . . . 215
 Faces of acute diarrhoea . . . 200
 — adenoic . . . 118
 — typhoid, atrophic . . . 265
 Face-scaraphe-fronsal hypothesis . . . 504
 Facial furia in tuberculous peri-
 tonitis . . . 118
 Facial attack, diagnosis from
 post and . . . 404
 Facial flaccidity, recurrent . . . 318
 — nervous diseases . . . 478
 Facial history in diagnosis . . . 4
 — predisposition to rheumatism . . . 145
 — acute and periodic parotitis . . . 481
 Facial response in prognosis of
 acute poliomyelitis . . . 411
 Fat, addition to milk of . . . 38
 — deficiency of causes in milk . . . 75
 — deficient in causation of rickets . . . 62
 — decomposition in milk, causes
 — lactic fat . . . 34
 — indigestion . . . 44
 — treatment of . . . 53
 — percentage in human and cow's
 milk . . . 71
 Fatty degeneration of the liver . . . 113
 Facial complications of scarletina . . . 127, 216
 Facial diptheria . . . 218
 — diagnosis from streptococci . . . 214
 Fecile-mucoid, elevation of . . . 548
 Feeding after the sixth month . . . 44
 — artificial . . . 35
 — frequency and quantity . . . 33
 — breast, management of . . . 15
 — by crowded milk . . . 33
 — diluted milk . . . 16
 — excessive volume in . . . 43
 — of healthy children . . . 25
 — infants in different cases . . . 31
 — from one to five years . . . 40
 — in relation to acute diarrhoea . . . 242
 — summary of methods of . . . 36
 — of syphilitic children . . . 342
 Fevers, advance in diagnosis of
 pyrexia, streptococci . . . 435
 Ferrous in rickets . . . 71
 Fever of acute rheumatism . . . 150
 Fevers, infectious . . . 122
 Fibrinous bronchitis . . . 151
 Fibrous, pulmonary . . . 152
 Fingers, curling in cases of
 Erb's palsy, children, congenital
 cerebral diplegia in . . . 474
 — pyrexia, streptococci . . . 264
 Fingers in inherited syphilis . . . 181
 Fists in tuberculous parotitis . . . 110
 Flaccidity in botuloid adults . . . 68
 Flaxseed, action in epidemic enteric
 febrile fever . . . 202
 Fluid, diagnosis of in empyema . . . 102
 Fluid, endocarditis . . . 364
 — rickets . . . 400
 Flows, infection with syphilis of . . . 218
 Follicular tonsillitis . . . 345
 — diagnosis of diptheria from . . . 218
 Fontanelle, anterior, development of . . . 10
 — importance of condition of . . . 3
 — bulging, in tuberculous meningitis . . . 121
 Fontanelles, condition of in meningitis . . . 100
 — abnormalities in rickets . . . 61
 Food, as addition to cow's milk . . . 34
 — parent . . . 33
 — as substitute for cow's milk . . . 33
 Foodborne's spots . . . 235
 Foreign body in bronchus . . . 152, 155, 217
 — larynx . . . 151, 157, 158, 218
 — nose . . . 122, 214
 — nasopharynx . . . 155, 212
 Foreign in dysphagia . . . 212
 Foreign in tracheitis . . . 118
 Fourth disease . . . 524
 Fracture osseum . . . 455
 Frase food, petting, composition of . . . 35
 Fraxinella's ataxia . . . 428
 — age-related of . . . 7
 "Frog-child" . . . 165
 Functional nervous disorders . . . 445
 Functional parotitis . . . 486
 Funnel chest . . . 8
 — following whooping-cough . . . 143

	1898		1898
Knee-jerk in diagnosis	45	Liver, catheter of	317
— importance of examination after		— disease of	312
typhoid	125	— displacement and malformation	312
— in pericardial pneumonia	30	— examination of	312
Koplik's spots	429	— fatty degeneration of	313
L		— functional derangements	320
AUC of progress in bottle and		— symptoms in primary splen-	
lactated children	37, 55	angia	151
Lactation, deficiency, to supply	47	Lumbar puncture in acute nephritis	165
Lactation, mother's diet during	46	— acute poly-encephalomyelitis	122, 123
Lactose, addition to milk of	48	— chronic	132
— percentage in human and cow's		— diagnosis	135
milk	21	— of intracranial abscess	437
Lak Lak, a dried milk, in after-		— of pneumococcal meningitis	121
treatment of diarrhoea	491	— of renal pelvis	155
— — — indication	48	— of pneumonia	41
Latching	490	— method of performing	42
Lactinase, deficiency, congenital	501	— in posterior bone meningitis	157, 200
Lambert's paralysis	494	— therapeutic results	47
Laryngeal dysphonia	216	— in tubercular meningitis	124
— chronic, congenital	512	— sleeping-sickness	250
Laryngismus stridulus	198, 490	Integ. sheaths in	153
— diagnosis of peritonsillar abscess	440	— chronic, character of eruptions	331
Laryngitis, acute catarrhal	351	— collapse of	140
— chronic	352	Integ. compression of in angina	203
— of larynx, syphilis	478	— disease of	192
— measles	514	— examination of	0
— membranous	110	— hæmorrhage from	191
— spasmodic	355	— intertrigo (see "Tuberculosis,	
— subglottic	123	pulmonary")	120
Laryngoscopy	118	Lymphadenoma	421
Larynx, disease of	354	Lymphatic glands, disease of	421
— examination of	44	— glandular enlargement, charac-	
— foreign bodies in	513	teristic groups	254
— new growths in	312	— leukæmic, acute	427
Late in delayed union	74	Lymphatics	425
Lateral chorda	105, 447	M	
Grange in acid dyspepsia	222	MACHON RHEUMATISM, diagnosis of	
— acute diarrhoea	201	Lachryphalus lani	441
— gastric	211	Male sex in treatment of typh-	
— relation of stomach	206	oid	110
— gastric and renal methods of	412	Malignancy of heart	191
— in pyloric region	175	Malignancies associated with	
— delay	439	syphilis	151
Laser's strategy	484	— of kidneys	334
Leeches in catarrhal laryngitis	514	— liver	142
— value of in pyæmia	50	— vesiculae	259
Legals test for leucæmia	75	Malignant disease of the kidney	190
Length, average at birth, etc.	18	— first	169
Leucocytemia in pneumonia and		— endocarditis	186
infundibula	90	— uterus	253
— description	120	Malaria, acute, kind, preference	
— rheumatoid arthritis	571	compensation in	53
Leukæmia, acute leucæmia	613	Marietta, thyroiditis	435
— spleno-megaly	413	Marietta	190
Leukæmia test for leucæmia	75	— methods of leucæmo-pneumonia	345
Leucorrhœa	599	— significance of eruptions in	429
Uterine, characteristic movements		— syphilis	150
in Africa	104	Massage, abdominal, in fractured	
— signs of disease in	7	children	25
Line-water as solvent of milk	38	— in acute poly-encephalitis	218
— preparation of	370	— position	227
Linzer's apparatus in leucæmia			
diagnosis	224		
Linking	466		
Liver affections in leucæmocytemia	585		

	PAGE		PAGE
Milk, rack, postmortem in, in autopsies	45	Myocarditis	506
— — — for normal infants	34	— clinical varieties of	504
— digested, in card-indigestion	45	— etiology and symptomatology	504
— dried, feeding by	34	Myositis ossificans	506
— effect of prolonged feeding on	47		
— in the etiology of subcutaneous	51	N AILS, affections of, in children	155
— food substances for	51	Nasal catarrh, acute	171
— basis, composition	51	— — chronic	174
— hardened, in card-indigestion	47	— diphtheria	175
— methods of preparing modified	50	— discharge, syphilitic origin of	173
— modified, in card-indigestion	50	— feeding, method of	172
— putrefaction of	51	— — in pharyngeal stenosis	171
— performed, in card-indigestion	45	— symptoms of infectious syphilis	172
— sealing	52	Nasopharyngeal obstruction in etiology of asthma	165, 166
— sterilization	52	Neural food, percentage composition of	55
— — in relation to acute diarrhea	504		
Milk food, percentage composition of	55	Nephritis, acute, diagnosis, prognosis, and treatment	147
Mucosarium and syphilis	175	— — etiology and symptoms	145
Mitral regurgitation	159	— chronic interstitial	145
— atrophic	159	— — parenchymatous	145
— development of	159	— complicating marasmus	147, 149
Mongolian idiosy	499	— locus of acute diarrhoea	254
— — association with cardiac malformation	151	— simulating	254
— — and cretinism, diagnosis between	155	— pneumococcal	144
Mongolian, appearance of finger in Meckel's sac ("Meckel")	117	— syphilitic	147
Morbus cerebri and morbus cerebri	365	Nerve fibres in asthma	166
— morbus	285	Nervous causes of constipation	151
— morbus Warfieldi	415	— — cerebral	160
Morgan's barrier in the etiology of diarrhea	456	— — vomiting	160
Morphine, rules for administering	513	— complications of diphtheria	141, 144
Moseley's food, percentage composition of	51	— — whooping-cough	147
Mother, hygiene of the nursing	56	— cough	145
Mother, power of fluids, tests for	55	— diseases, acquired	145
— symptoms of chorea	193	— — bacterial	145
Mouth, diseases of	195	— — cancer	145
Mucosa disease	194	— diseases associated with syphilis	145
Mucus in vomit, in diagnosis of pyloric stenosis	185	— — functional	145
Mumps, etiology	137	— symptoms of acute nephritis	166, 167
Mumps, dates of incubation	137	— — adenoids	166
— parotitis, etc	137, 151	— — chorea	166
— etiology and symptomatology	151	— — hydrocephalus	166
Mucosa, diseases of	506	— — in relation to various	166
Muscular atrophy, peracute type of development, prognosis	417	— — in rheumatism	166
— distally	400	— systemic diseases of	167
— pain in rheumatism	197	— — effect of rickets on	166
— symptoms in chorea	194	— — examination of	166
— system, effect of rickets on	67	— — infectious of	166
Mutual bath, method of preparing	519	— — syphilis of	166
Myasthenia gravis	504	— — tuberculosis of	167
Myocarditis, acute, chronic, method anatomy	376	Nesbitt's condensed milk	54
— — — prognosis and treatment	376	Neuroasthma with cerebral disease	166
— — — symptomatology	376	Neuritis	166
— etiology of	161	Neuritic muscular atrophy	406
— subacute	380	Neuritis of diphtheria	161, 163
— pneumococcal	129	— peripheral	404
		— — diagnosis of acute pain	166
		— — myelitis	166
		Neuroticism as cause of heart and pylorus	166
		Neuropathic factors in chorea	166
		— substances	166
		— — and asthma	166

- Neurotic child, the 445
 — Impairment in atiology of this 455
 — — night terrors due to 456
 New-born, jaundice in the 514
 New growth of larynx 417
 Nightmares 456
 Night sweats in pulmonary tuberculosis 751
 Night terrors 456
 Nipples, care of in breast feeding 46
 Nocturnal enuresis 457
 Nodal fever 474
 Nodes of rheumatoid arthritis 475
 — subcutaneous in rheumatism 475
 Noma podagii 459
 Nose, diseases of 122
 Nursing, contraindications to 12
 — infections 147
 — period, mother's diet during 28
 Nystagmus in acute poliomyelitis 614
 — congenital cerebellar ataxia 477
 — Friedreich's ataxia 478
 — with head-rolling 484
 — in inherited syphilis 101
O
OBESITY, precocious 418
 Obstetrical palsy 471
 Obstruction near Meckel's diverticulum 501
 Ocular complications of measles 212
 — symptoms of sclerotal sarcoma 471
 — chorea 464
 — syphilis of nervous system 456, 491
 — — tuberculosis meningitis 125, 128
 Odema 494
 — in acute syphilis 497
 — of eyelids 494, 497
 — — in rheumatic pericarditis 174
 — glottis 497
 — neonatorum 494
 Ophthalmograph, method of 514
 Ophthalmic diseases of 255
 Optic atrophy in etiology of thobis 251
 Optic atrophy in inherited syphilis 481
 Operation in pyrexia venosa, indications for 259
 Ophthalmia neonatorum 269
 — pneumococcal 195
 Ophthalmoscopic examination 51
 — — importance in diagnosis of syphilis 199
 Ophthalmoscopy in congenital syphilis 485
 — posterior basic meningitis 157
 Optic or rheumatic pericarditis 175
 — rules for administration 511
 Oppenheim's paralysis 504
 Opsian index in Bier's treatment of arthritis 174
 — — meningitis 194
 — — pulmonary tuberculosis 154
 Optic atrophy, hereditary 482
 — — and neuritis due to intracranial tumours 491
 Optic due to health and disease 51
 — results in diagnosis of typhoid 466
 — — rarity of, in infants 51
 Ovario-compensating rumps 168, 169
 — in inherited syphilis 168, 169
 Organic nervous diseases 479
 Ossification, defective 467
 Ovarian dysgenesis imperfecta 468
 Othinosauria 21, 795
 Otorrhoea, diagnosis of source from 79
 — due to pneumococcus 115
 — simulating typhoid 444
 Otos media compensating scarlatina 127
 — — diagnosis of pneumococcal meningitis from 111
 — — diagnosis of pneumonia from 35
 — in inherited syphilis 162
 — — pneumococcal 112
 — — simulating typhoid 469
 — — strept. throatitis due to 485
 Ovarian tumour raising prostration development 428
 Ovario-compensating rumps 151
 Oxycephaly 498
 Oxygens in pneumonia 106
 Oxyuris vermicularis 506
P
PACK, not, in chorea 266
 — — Pass as symptoms of rheumatism 161
 Palate, perforation of, in inherited syphilis 476
 Palpation in diagnosis of acute rheoditis 493
 — method of 5
 Palpus, angusted central 471
 — congenital cerebral 471
 — obstetrical 471
 Pancarditis, rheumatic 179
 Papilloma of larynx 137
 Paralysis 480, 471
 Paralytic conditions simulating 455
 — Erb's 479
 — fixed 175, 175, 504
 — hardy spasm and tremor 481
 — hysterical 486
 — juvenile general 183
 — Landry's 188
 — neonatal, in chorea 184
 — Oppenheim's 184
 — post-diphtheritic 141, 141
 — test of motor power of limbs in 51
 — Weirlog-Hoffmann 482
 Paralytic symptoms of acute poliomyelitis 147
 — — tuberculous meningitis 141
 Paraplegia, spastic, examination of spine in 5
 Parasites, intestinal 496
 Parasitic stomatitis 255
 Paratyphoid infection 206
 Paratyphoid nephritis, chronic 205
 Paratyphoid, acute 218
 — specific (see "Meningitis") 211
 Patient's notes in inherited syphilis 194

	184		185
Tarlet's nodes in children	15	Thoma, theories of	260
Tea, hyperemia, treatment in		— Blackwell diagnosis of (1941)	264
thrombosed anastomosis	174	sore throat	264
Telemetering of milk	11	Throat, infection, collapse of lung in	310
Telesar index in throat	445	Throat, dry	260
Telnet (acute pharyngitis)	155	— pseudotuberculous	166
— lacunar stage	155	— rheumatic	152
— infant form	55	— with acute edema	260
— median syndromal	155	— interictal	112
Telnet, chronic	152	Thrombopar in pneumonia	30
— infection	150	Thrombopar in pneumonia	
Thrombopar causing erythema		diplegia	120
lating anastomosis	154	Thrombotic diseases	115
Thrombotic diseases	152, 110	— arterial	115
Thrombotic, syphilitic	170	— embolic	115
Thrombotic	111	— endocarditis	260
Thrombotic risk in acute edema	45	— heart disease	117
— method of preparation	110	— infection the	50
Thrombotic, method of	3	— infection, spinal enlargement	111
Thrombotic, acute thrombotic pharynx		— anastomosis	110
— signs, etc.	171	— myocardial	110
— prognosis and treatment	170	— nephritis	114
— symptomatology	171	— oral cavity	111
— history of	167	— pericarditis, partial distal	
— pseudocystic	167	— and chronic	110
— tuberculous	110	— pyogenic and treatment	110
Thrombotic, adherent	110	— lymphatic	111
Thrombotic, parathyroid, family	111	— pericarditis	111
— treatment	5	— pharynx	110
Thrombotic, pharynx	110	Thrombotic in theiology of	
— symptoms, syphilitic	111	— anastomosis	111
Thrombotic, syphilitic	111	— acute	117
Thrombotic, syphilitic	111	— chronic	110
— diagnosis of acute pharynx	110	— congestive heart	111
— syphilitic	111	— diagnosis of chronic heart	111
— treatment	110	— emphysema from	111
Thrombotic, acute, of chronic	110	— pericarditis from	111
— treatment	110	— primary and congestive	111
Thrombotic, diseases of	110	— typical form	111
Thrombotic	111	— dilation of stomach in	111
— pseudocystic	111	— emphysema following	111
— syphilitic	110	— infection	110
— tuberculous	110	— intestinal thrombotic	111
Thrombotic, chronic	111	— myocardial, complications	
Thrombotic type of myocardial atrophy	110	— and response	11
Thrombotic (see "Whispering-cough")	110	— acute	11
Thrombotic	111	— chronic	11
Thrombotic, chronic, of chronic	110	— diagnosis	11
— signs, etc.	111	— etiology	11
Thrombotic, chronic, of chronic	110	— prognosis	11
— signs, etc.	111	— symptomatology	11
Thrombotic, chronic, of chronic	110	— treatment	11
— signs, etc.	111	— primary	11
— treatment	110	— secondary, non-spreading	11
— prognosis	111	— pulmonary disease following	111
— symptomatology	111	— gangrene in	111
— treatment	110	— rheumatic	111
— primary	111	— interictal	111
— secondary, non-spreading	111	Thrombotic (complication) of (111)	
— pulmonary disease following	111	— acute	111
— gangrene in	111	— whispering-cough	111
— rheumatic	111	Thrombotic, diagnosis of emphysema	111
— interictal	111	Thrombotic, acute (see also "Whispering-cough")	111
Thrombotic (complication) of (111)			
— acute	111		
— whispering-cough	111		

	Index		Index
Poli-morphous cellitis	204	Pulmonary complications of dys-	
— acute, bacteriology and medical		beria	255, 261
anatomy	212	Etiology	259
— constitutional symptoms of	213	pathology	257
etiology	211	signs in pneumococcal pneu-	
Potomacitis, acute, complications,		monia	31
diagnosis, and prognosis	215	— secondary, leucopenia	
— paralytic symptoms of	217	rashes	147
— typhoid	219	stenosis, congenital	255
age-incidence of	2	typhoid	279
toxic	216	— and thoracic valve lesions	135
ventricular fluid in	25	tuberculous	250
diagnosis of symptoms	214	— causes of mortality in	260
physic from	152	Pulse rate in infancy	71
Pulmonary emphysema	432	interventricular aneurysm	331
Pylorus	502	Punctate chest in diagnosis of	
— in chronic interstitial nephritis	594	scarletina	167
Pyloric stenosis	515	Pyloric signs in rhoma	252, 260
— history	501	Pyrexia in acute diarrhoea	200
Pyloric tube, procedure regarding	516	— diagnosis of intracranial lesion	234
Pseudo-epitheliomatous	52	— Erysipelas	416, 425
Pseudobubonic meningitis	545	— hemorrhages	430
— paratuberculous	545, 544	— infective	430
— — diagnosis from acute poly-		— in neuroleptocystic meningitis	205
myelitis	233	— rheumatic	332
Pyloric tube aneurysm	161	— symptomatic and essential	165
— age-incidence of	2	Pyrexia, toxæmic in acute lymphatic	
— in older children	163	leukæmia	427
Pyrexia, toxæmic in acute rheu-		Purulent penicillitis	102
matism	136	Pyrexia, acute	407
— in children	164	— diagnosis of pneumonia from	37
Pyloric, dilatation, history, pro-		pyelocystitis	421, 422
cedure	58, 60	— diagnosis of typhoid from	294
Pyloric tube, method of preparing	512	Pyrophosphoric, evaporative	120
Pyrexia, in rheumatoid arthritis	174	Pyrene spores	321
Pyrexia, in scarlet fever development	272	— diagnosis of hypertrophic	
— death	265	stomach from	205
Pyrexia, lentic and typhoid	476	— stenosis, age-incidence of	7, 265
Primary pneumonia (see "Pneu-		— — diagnosis from acid dyspepsia	271
monia, pneumococcal")	30	— — of Herpeszoster infection	334
Pyrexia, low	477	— — and prognosis	265
Pyrexia, associated atrophy		— — dilatation of stomach in	274
diagnosis from pyrexia	471	— etiology and medical anatomy	264
Pyrexia in scarlet	22	— physical signs	266
Pyrexia, defervent, distress in work	38	— symptomatology	268
— percentage in human and cow's		— treatment	269
milk	23	Pyrocarditis	365
Pyrexia, paratuberculous	51	Pyrosis	311
Pyrexia, due to thermal causes	166		
Pseudo-hypertrophic myopathy	192	QUARANTINE in acute forms	221
— age-incidence of	7	— Diphtheria	224, 235
Pseudobubonic, (typhoid-like)	477	— measles	232, 239
— infection from Eberth's aneurysm	408	— mumps	232, 233
Pseudo-paratuberculous	495	— rubella	225, 233
— test of water-power of fluids in	55	— scarletina	221, 224
Pseudo-reduplication of second		— varicella	231, 240
spinal sound of heart	153	— whooping-cough	231, 251
Pyrexia, see	455	Quarantine in acute rheumatism	250
Pyloric stenosis	515	Quincy	327
— diagnosis of symptoms from	160		
— adhesion in infants	58		
— collapse	534		
— — diagnosis in rheumatism	542		
— — esophagus following	534		
— complications of aneurysm with	257		

QUARANTINE in acute forms	221
— Diphtheria	224, 235
— measles	232, 239
— mumps	232, 233
— rubella	225, 233
— scarletina	221, 224
— varicella	231, 240
— whooping-cough	231, 251
Quarantine in acute rheumatism	250
Quincy	327

RACHITIS (see "Rickets")	51
Risk of rickets-pox	281
— rickets-like lesions	275
— measles	231

East of umbella	440	Rheumatism, chronic in	168
— scutellaria	440	— etiology	168
Eases, differential diagnosis of	225	— in etiology of leucocytes	440
— of influenza	265	— in	455
— syphilis	170	— general appearance in	6
Raw meat juice in cardiodigestion	50	— latent chronic in	251
— — — and pulp, methods of preparing	171	— post-vascular	145, 147
Ranuncul's disease, hemoglobinuria associated with	191	— relation of rheumatoid arthritis to	171, 173
Reflux irrigation in acute diarrhea	247, 249	— symptomatology	170
— — — chronic diarrhea	247	— treatment	171
— — — lavage, method of	247	Rheumatoid arthritis, etiology	170
Red measles, the	415	— exact anatomy, diagnosis, and prognosis	171
Re-education in convalescence from cholera	270	— — — symptomatology	170
Recurrent vomiting	81	— treatment	171
Reflex irritation and convulsions	454	Rheumatic prostration, acute	26
Reflexes in cholera	407	Rhachis, chronic	145
— — — irritation of	11	— metabolism	215
— — — in Friedreich's ataxia	478	Rheumatism complicating measles	232
— — — myopathy	500	Rhachis	171
Renal calculus	400	Rib insertion for empyema	166
— complications of dysentery	218	— ribs, affected in, in rickets	64
— disease	394	Rice-water in diluent of milk	38
— — — malignant	397	Rickets, acute (see "Scurvy")	73
— — — pelvis, dilatation associated with pyloric disease	262	— in variation in leucocytes	112
— — — tuberculosis	143	— — — diarrhea	127
Residual cholera	165	— congenital (see "Congenital hyperthermia")	72, 467
Respiration-rate in tuberculosis meningitis	124	— diagnosis	79
Respiratory distress pending collapse of lung	348	— — — of hydrocephalus, brain	100
— symptoms in communicative broncho-pneumonia	347	— — — subconjunctival hyperemia from dilatation of stomach in	174
— — — influenza	368	— etiology in	50
— — — toxemia, diseases of	313	— morphology of bronchio-pneumonia	348
— — — effects of toxins on	318	— — — of measles	418
— — — examination of	5	— spinal (see "Aerodysphagia")	66
— — — cyphosis of	178	— late or delayed	74
— — — tuberculosis of	128	— method anatomy of	50
Retropharyngeal abscess	317	— nervous disorders associated with	418
Rheumatic anemia	143, 445	— predisposing to anæmia	113
Rheumatic endocarditis, acute	278	— — — to after-effects of typhoid	143
— cystitis	112	— progress	20
— factor in cholera	117	— species treated in	174
— heart disease	367	— symptomatology of	10
— infection, the	144	— treatment	21
— manifestations in cholera	102	Rhizy, chest, appearance of	5
— nodules	145	— hypotonia	16
— pericarditis, acute	273	— osary	64
— peritonitis	313	Rick's food percentage composition of	35
— pleurisy	152	Russett, both for	15
— pneumonia	152	Rick's biscuits, percentage composition of	35
— purpura	152	— whole milk food percentage composition of	35
— risk simulating acutania	68	Robinson's patent barley, percentage composition of	35
— trachitis	315	Rubella (see "Rubella")	211
Rheumatism, acute, age-incidence of	7	Roundworm	107
— bacteriology of	143	Rubella, stages of incubation, quarantine, etc.	221, 111
— cardiac	353	— diagnosis from scutellaria	228
— as cause of headache	463	— etiology and symptomatology	231
— characteristics in children	142	Russett's grain in malition meningitis	100
— acute and prognosis	143		
— diagnosis	152		

	Page		Page
SABRE, tilia of inherited syphilis	183	Seasonal incidence of measles	221, 236
Sachs's disease	344	— — — groups	271, 331
Saddle-sore of inherited syphilis	474	— — — posterior lobe meningitis	300
Saltan, E.	438	— — — rheumatism	243
Salivary glands in acute rheumatic heart disease	479, 485	— — — rubella	221, 253
Salivates in chorea	168	— — — scarlatina	221, 254
— specific action in rheumatism	154	— — — varicella	243, 254
Salivary anal poisoning	84, 153	— — — of Vincent's angina	337
Salivary apertures	479, 480	— — — whooping-cough	221, 248
— infection in acute diarrhoea	461	Secondary broncho-pneumonia	144
— injection in acute syphilis	168	Sedatives in chorea	166
Salivary glands, diseases of	255	Serbi	279
Salt percentages in human and cow's milk	11	— — — foods, methods of preparing	
Santonin in treatment of round-worms	385	— — — infusion of	511
Sarcosis, adeno-	144	Septic complications of scarlatina	243
— of kidney	191	— — — embolectomy	254
— liver	204	— — — parotitis	258
Savory & Moore's food, percentage composition of	55	— — — retropharyngeal abscess	139
Scaling of milk	31	Serous, multiple	142
Seaming, speech	468, 477	Serous diagnosis of meningitis	104
— in cerebellar encephalitis	224	— — — test in inherited syphilis	192
Scarlatina, complications and aspects of	177	— — — diagnosis of typhoid	304
— — — dates of incubation, quarantine, etc.	223, 224	— — — therapy in acute rheumatism	158
— — — diagnosis	225	— — — diphtheria	243
— — — of pneumonia from	34	— — — epidemic cerebrospinal fever	262
— — — etiology	144	— — — larval diphtheria	218
— in etiology of chorea	258	— — — post-toxic meningitis	206
— following diphtheria	145	Sex-incidence of acid dyspepsia	271, 272
— prognosis and treatment	229	— — — acute poly-morpho-nucleitis	219
— symptomatology	125	— — — primary peritonitis	204
— variations of	426	— — — abscesses	184
Scarlatiform rash of rubella	254	— — — bacillaria	201
Scarring round mouth and arm as sign of inherited syphilis	6	— — — chorea	148
Schick's disease	419	— — — erythema nodosum	173
Scout-legged progression	475	— — — hydrocephalus atonia	379
Sclerosis, cerebral	474	— — — lymphoplasm polio-encephalitis	200, 222
— disseminated	431	— — — intussusception	302
Schistosom (see "Scurvy")	75	— — — pericerebral peritonitis	171
Schismata, causes of	455	— — — pseudo-hypertrophic atrophy	201
— — — colic and other causes of	280	— — — rheumatism	145
— in diagnosis of chorea	415	Sexual organs, precocious development of	422
— indications of	65	Sight, loss of	34
Scurvy, age-incidence of	7	Single atrophic myopathy	391
— — — diagnosis	76	Slush, thrombosis of	485
— — — of erythritic epiphysis from	187	Skeletal systems, evidence of disease in	7
— etiology	71	Skin, edema in erysipelas	143
— marked analgesia	78	— — — diagnosis of enlarged thyroids	428
— prognosis	74	— — — pneumonia and tuberculosis	57
— symptomatology	75	— — — parietal pneumonia	197
— treatment	74	— — — renal tubules	184
Seasonal incidence of acute enteric fever	221	— — — rheumatoid arthritis	475, 476
— — — acute poly-encephalomyelitis	211	— — — vesical calculus	466
— — — chorea	445	Skin alterations of inherited syphilis	124
— — — diphtheria	223, 235	— — — changes in acute diarrhoea	394
— — — epidemic cerebrospinal meningitis	264	— — — rheumatoid arthritis	172
— — — erythema nodosum	175	— — — manifestations of meningococcal meningitis	201
— — — lymphogranuloma	301	— — — rheumatism	152
		— — — purpuric spots in scurvy	38
		— — — thickening in scarlatina	223
		Soft, abnormalities in osseous	84
		— — — syphilis	181
		— — — symmetry of, in adenal carcinoma	214

	1954		1954
Shall, thermometers of	20	Sheathed milk in pyothorax of	
— reservoir of disease in	7	tuberculosis	118
Sheep-sucking	151	Sheep-sucking wounds, treatment of	171
Shedder	125	Shed, application of	154, 155
Shedder using nasal chalice	125	Shed's disease	129
Sodium bicarbonate in diet of		Stomach capacity of goats	31
calf	31	— dilation of	134
— citrate, addition to milk of	71	— disease of	293
Somnolence	140	cytomegalic thymus of	174
Sore throat as symptom of disease		Stomatitis in dairy animals	121
milk	145	— clinical varieties of	125
Spasms, corporeal	145	— gangrenous	127
— in the treatment of pyothorax	154, 155	— symptoms and treatment	295
— polio	271	— lymphatic	131
Sporadic lameness	131	Stomach, examination of	31
Sporous system	154	— rumen contents, etc.	31
Sputa, investigation	150	Striker, rumenitis bacterial	134
— bloody	145	Struthius, rules for administration	121
— purulent, healthy	151	Strep in chorio	151
Speech, alteration in throat	151	Strutling	139
— defective	151	Subconjunctival hemorrhage in	
— delayed development of	152	sheep-pastoral	147
— development of	22	Subcutaneous abscess, method of	112
— in adult	153	— nodules in formation	145
— in child	152	Sublingual abscess	127
— loss of	153	— in sheep-pastoral	147
— occurring in umbilical cord		Subperitoneal abscess, diagnosis of	
pneumia	211	— lameness in dairy	71
— congenital umbilical abscess	127	Substitutes for cow's milk	53
— Frederick's abscess	124	Sugar, addition to milk of	38
— diphtheria	145	Substratum and its dissection	122
Suicidal and changes in Frederick's		Suppurative pyothorax	200
abscess	124	Suppurative abscess, varieties of	111
— pyothorax abscess	124	Surgical treatment of pyothorax	
— treatment of abscess	56	abscess	200
Suicidal, examination of	5	Swath, in thermometers	151
Suicidal, disease of	127	Swathed and unswathed cow	
— displacement and enlargement of	121	disease cases	50
— examination of	3	Swathing of eyelids in dairy	27
Synovectomy for primary synovitis		— in dairy	26
Synovitis, primary	134	Synovitis of chorio	157
— of joints	135	Symptoms, carrying import in this	
— employment in diagnosis osteoarthritis	137	disease and ability	1
— disease of articular	125	Synovitis, degree of, in pyothorax	200
— synovitis	135	Synovitis, synovitis	134
— tuberculous pyothorax	144	Synovitis and acute excretion	135
— von Thomsen's abscess	125	— of the alimentary system	134
Synovitis, pyothorax	125	— bones and joints	134
— of von Thomsen's disease from	125	— cardiovascular system	137
Synovitis, when to start	44	— etiology of synovitis	132
Synovitis, treatment of, in tuberculosis	131	— of lameness	131
Synovitis, when to start	44	— of the gastro-intestinal system	137
Synovitis, treatment of, in tuberculosis	131	— hereditary	131
Synovitis, treatment of, in tuberculosis	131	— inherited, diagnosis, prognosis, and treatment	132
Synovitis, treatment of, in tuberculosis	131	— muscular and infective	132
Synovitis, treatment of, in tuberculosis	131	— mode of infection	130
Synovitis, treatment of, in tuberculosis	131	— symptoms	130
Synovitis, treatment of, in tuberculosis	131	— subcutaneous, cerebrospinal fluid	25
Synovitis, treatment of, in tuberculosis	131	— and acute disease	131
Synovitis, treatment of, in tuberculosis	131	— of the nervous system, etc.	130
Synovitis, treatment of, in tuberculosis	131	— respiratory system	130
Synovitis, treatment of, in tuberculosis	131	— and rheumatoid arthritis	131
Synovitis, treatment of, in tuberculosis	131	— swelling synovitis	132, 133

- Syphilis of the skin, etc. 379
 — splenic infarct and glomer. 380
 Syphilitic calciosis of liver: diag-
 nosis of carcinoma from 381
 — endarteritis, thrombotic imma-
 turing, acute polymorpho-
 litic 382
 — syphilis, age-incidence of 3
 — diagnosis of scarlet from 79
 — mental degeneration 143
 — test in pathology of liver 382
 — see 384
 Syringomyelia 358
- T**
 TABES, diagnosis of Fried-
 reich's ataxia from 430
 — hematuria 341
 Tache fibrilante in pericardial
 pneumonia 369, 371
 — in tuberculous meningitis 425
 Tachycardia 355
 Tachypnea, diagnostic sign of 30
 — in pericardial pneumonia 369
 Tactile 399
 Telling, storage: age of contusion
 37
 Tape-worm 368
 Tartarized stye, preparation of 366
 Teeth, choice of 36
 Teeth, average of eruption of 35
 — cancer 358
 — causing symptomatic anæmia
 e.g. malaria 354
 — decayed and faulty, in rickets 36
 — examination in diagnosis 3
 — growing 361
 — in inherited syphilis 478
 Telling, association of contus-
 ions with 432
 Temperament in acute bronchitis 347
 — acute follicular tonsillitis 369
 — chorea 364
 — consecutive broncho-pneumonia 347
 — diphtheria 337
 — pneumococcal pneumonia 34
 — traumatic infection, effect of
 salicylates on 354
 — rheumatism 359
 — scarlatina 365
 — infectious meningitis 344
 Tetanus in rickets 36
 — strychnine 75
 Tetanospasms in chorea 463
 Tendon reflexes 35
 Tetanussy in acute poliomyelitis 221
 Tertiary affections in inherited
 syphilis 463
 — tumour causing posterior
 development 495
 Tetany 445
 Thalamic microphallid 220
 Therapeutic measures 321
 Thromboses 366
 — as cause of angina 468
 Thromb, cardiac, examination for 31
 Throat affections in rheumatism 748
 Throat emphysema of scarlatina 377, 379
 — rough 388
 — sore 395
 — symptoms of diphtheria 337
 — tubercle 393
 Thrombosis, diagnosis of acute
 poly-arteritis from 417
 — as etiology of hemiplegia 404
 — venous stasis 406
 Thrombi 353
 Thoracic asthma 417
 Thymic enlargement of tape-worm 368
 Thymus, enlargement of
 — weight in health and disease 414
 Thymic deficiency and tetanus 414
 — enlargement in chorea 364
 — extract in rheumatic arthritis 328
 — as treatment of tetanus 444
 Tinea, mycotic dermatitis of 363
 Ties 435
 Tissue, how to examine
 — mycotic affections of 365
 Tissuefactory after scarlatina 336
 Tonsillitis, acute follicular 351
 — chronic 358
 — diagnosis of diphtheria from 338
 — pneumococcus in etiology of 346
 — recurrent 346
 Tonsils, diseases of 355
 — enlarged, as cause of tetanus 468
 — and adenoids 325
 — predisposing to asthma 369
 — in strychnine poisoning 449
 — as cause of hemiplegia 444
 — tuberculous of 328
 Top-note, finding 35
 — apparatus of use for 360
 Tracheitis resulting from herpes
 tonsil of metastomatid 470
 — with retropharyngeal abscess 341
 — rhinorrhoea 347
 Tracheal 369
 Transfusion in acute nephritis 368
 Traumatic injury 441
 Tremor, acute, in acute poly-arteri-
 tides 335
 Tremor, and pulmonary water
 lesions 385
 Tremor in chorea 360
 Tremor's sign in tetany 449
 Tubercles, choroidal 74
 Tubercles, tests in pulmonary
 tuberculosis 416
 — in treatment 350
 Tuberculosis, abdominal diagnosis
 of achilia from 385
 — abdominal, diagnosis of He-
 nrich's parapsia from 460
 — treatment 348
 — age-incidence of 316
 — appearance of hands in 3
 — in radiation of rheumatic arthritis 387
 — of the respiratory system 316
 — diagnosis from bronchiectasis 352

	PAGE		PAGE
Tuberculosis, diagnosis of various		Tumors, infectious intracranial	142
— by bacteriopsentia from	123	Typhoid and allied affections	285
— — empyema from	124	— condition of spleen in	172
— — pneumonia from	95	— diagnosis of constipation from	125
— — typhoid from	203	— — exanth from	165
— of the digestive system	115	— prognosis and diagnosis	288
— etiologic factors	120		
— in etiology of pleurisy	154	U	
— following whooping-cough	245	Ullrich, Gustav	275
— general appearance in	5	— — sublingual, in whooping-	
— — solitary, clinical types of	189	cough	247
— — — of lungs	124	Ulceration of mouth	175
— — remedial treatment	133	— — sublingual	177
— of the gastro-intestinal system	141	Ulcerative endocarditis	176
— liver	97	— — gastric	273
— — mechanical glands	143	— — stomatitis	155
— — secretory glands	142	Uremia	365
— modes of infection	115	Use and misuse	163
— of the nervous system	178	Urinary changes in pneumonia	54
— prophylaxis	147	Urine in childhood	59
— pulmonary	110	— — hyperacidity of	261
— — clinical varieties	121	— — metastasis of, in diagnosis of	
— — diagnosis	121	exanth	405
— — treatment	134	— — normal variation of	266
— renal	141	— — tests for azotemia in	75
— of the respiratory system	128	Cytruria accompanying pyuria	117
— and rheumatism, family inher-		Urethral tube, a cause of cough	111
itance of	145		
— circulating (spontaneous)	411	V	
— splenic enlargement in	413	Vaccine therapy in epidemic	
— of the tonsils and larynx	115	corrospinal fever	301
Tuberculosis and convulsive		— — — — — maligant reaction	
brachycephalus, diag.		— — — — —	355
both between	140	— — — — — post-basis meningitis	306
— crinia	144	Vaginitis	110
— endocarditis	135	Valvular disease, chronic, prog-	
— eulipia	141	nosis and treatment	183
— epistaxis	144	— — — — — various forms of	181
— infection, the	115	Vaccinia (see "Chicken-pox")	250
— intracranial tumors	117	— bulla, gangrenous, and hemor-	
— joint disease, diagnosis of acute	122	rhagea	151
polyseria from	129	— dates of incubation, quarantine,	
— mechanical factors, diagnosis of		etc.	241
perforated from	125	Vermis in acute nephritis	195
— mesangitis, cardiac irregularity		— — — — —	196
a sign in	188	Vesicae (see "thrombosis")	195
— — diagnosis	125	Vesical calculus	190
— — of abscess from	127	Vesicles of chicken-pox and small-	
— — etiology and method of action	121	pox	251
— — diagnosis	125	Vesicle's agent	127
— — symptoms and signs	122	Voice changes with adenoids	185
— — treatment	127	— — retropharyngeal abscess	114
— — and tumors, cerebrospinal		Vomiting in acute gastritis	273
fluid in	36	— — bilious infants	58
— pericarditis	135	— — bruised children	59
— peritonitis	124	— — colic, pericolic, or retracted	74
— — notes on it	125	— — diagnosis of pneumonia	
— — diagnosis from syphilis	186	from	55
— — liver	134	— — in distention of the stomach	278
— — pleurisy	132	— — of epidemic diarrhoea	199
— — retropharyngeal abscess	112	— — in functional degeneration of	
— — tumors, diagnosis of (see		liver	349
polyseria from)	141	— — primary tubercular	151
Tumors, malignant renal	259	— — pyloric stenosis	266
— — fibrotic in structure	256	— — tuberculous testicles	111
Tumors, intracranial	141	— — various classes of	456
		Vin Jaksch's anemia	409

	Page		Page
Von Jaubert's anemia, diagnosis of	416	Whey, preparation of	599
— spleen anemia from	416	White-wine whey, preparation of	599
— — diagnosis from typhoid	420	Whisk-whisk, feeding by	50
Von Plummer's test in pulmonary tuberculosis	134	Widal test in diagnosis of typhoid	205
Vulvovaginitis	400	Wieders's disease	392
— gonorrheal	406	Whooping-cough, consecutive	348
		— in children pneumonia of	348
		— see cough in	345
WALLING, average age of	92	— claim of incubation, quarantine	345, 345
Walnut bark	424	— — — — —	345
Waller's disease	404	— diptheria in throat in	8
Wassermann reaction (in Fleming's modification of)	194	— etiology	345
Wasting in etiology of hookworm pneumonia	347	— in etiology of hookworm	347
— pleuro-pneumonia	347	— sequelae and diagnosis	345
— syphilitic	344	— symptomatology and complications	347
Wasting	39	Wax's bottles in diagnosis	346
Wassermann, diagnosis of	194	Wax-shadows, compound	406
Weight, averages of both, etc.	15	Waxes, injection	305
— child	15		
Wieders-Hoffmann paralysis	450	YACHTING	11
Wetting	91	Yeast fungus in etiology of thrush	455
Whey and cream in bacteriology	60	Yellow atrophy, acute, of liver	320
— — — — —	91	Yon's (Brewer's) test in pulmonary tuberculosis	434
— in etiology of such	91		

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